MANUAL of MEDICAL EMERGENCIES

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DEDICATION

TO THE GENERAL PRACTITIONER
WHO IS EXPECTED
TO SEE ALL KNOW ALL AND DO ALL
IN THE FIELD OF MEDICINE
AND WHO TO HIS EVERLASTING CREDIT
FULFILLS THESE EXPECTATIONS
ADMIRABLY

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Preface to Third Edition

THE AUTHORS wish to emphasize that the objective of this manual is to make available to the physician a handy reference that will facilitate meeting acute emergencies We feel that unnecessary deaths occur because the physician is not aware of the necessity for or the methods by which acute interference with such fundamental processes as respiration and circulation can be eliminated The manual is not designed to provide complete information of a diagnostic or therapeutic nature it is designed primarily to help the physician save the patient s life in those first few critical minutes

In this revision the major changes have been made in the extension of the material on acute poisoning to meet the problems evoked by the introduction of new toxic drugs The list of common drugs and poisons has been extended to facilitate identification Some changes have been made in the problems of acute circulatory emergencies

We are again indebted to all those who have made suggestions and provided helpful criticism

Preface to First Edition

FREQUENTLY the physician is precipitated into an emergency situation in which he is compelled to institute immediate therapeutic as well as diagnostic measures and for which he may be unprepared This is particularly true of the general practitioner, whose ubiquitous practice of medicine demands an all-encompassing and practical working knowledge of internal medicine surgery pharmacology toxicology and the like

The urgency of the situation does not permit reflection leisurely search of the literature or consultation for assistance in solving the problem at hand It seemed therefore that a small manual in which concise, readily accessible and reasonably complete coverage of emergency situations with particular respect to immediate detailed therapeutic measures, would be useful to the busy practitioner

Furthermore the physician is concerned almost daily with problems in patient management, for the solution of which he is dependent on knowledge of practical details which are neglected or omitted from

most medical curricula As a consequence this manual deals with such problems as the sedation of the agitated patient the administration of oxygen therapy the care of the comatose patient and technics of venipuncture

It is emphasized that this manual is designed to cover only the acute emergency extensive diagnostic measures and continued definitive therapeutic measures are deliberately excluded The material is presented in the belief that many patients lives can be saved with appropriate emergency therapeutic drugs and procedures efficiently applied until complete diagnosis is possible and specific therapy is determined It can be expected that some of the ancient and revered emergency drugs and procedures will be either totally neglected or treated critically On the other hand pharmacologic and physiologic justification is established for many of the older remedies We have attempted throughout to confine recommended drugs equipment and procedures to those that are easily available transportable and workable

We are indebted to Dr H Russell Meyers for the introductory discussion of general principles of management and for Chapter 6 on Head Injuries to Dr L E January for his assistance with the chapters on care of comatose patients and circulatory emergencies to Dr Rubin Flocks for the material on care of the bladder in comatose patients to Dr Sidney Ziffren for the material on immediate treatment of patients with burns and to many other colleagues in and out of the College of Medicine of the State University of

Iowa who have given valuable advice Acknowledgment is due the authors and publisher for permission to reproduce Figure 22 from W E Nelson s *Textbook of Pediatrics* (4th ed Philadelphia W B Saunders Company 1946) and Figures 25 27 and Figure 29 from John S Lundy s *Clinical Anesthesia* (Philadelphia W B Saunders Company, 1942) In addition we are indebted to Miss Dorothy Skubal and Mrs Olive Bailey efficient and hard working secretaries to Mr Frederick Kent for his excellent photography and to the staff of the Year Book Publishers for unstinted help

General Principles in Emergency Treatment

UNDER the stress of dramatic unanticipated emergency medical situations the physician often unconsciously turns to diagnostic and therapeutic practices redolent of mysticism witchcraft and dogmatism The silent scrutiny of bystanders the supplication of relatives and the critical state of the patient provoke the adoption of attitudes and the application of diagnostic and therapeutic measures which the physician finds difficult to explain after he has had time to reflect upon the circumstances in which he found himself Many a patient has gone to his death or in slightly more favorable situations has suffered unnecessarily severe and prolonged morbidity because physicians attempting to solve emergency medical problems are unwilling and reluctant to apply or are unfamiliar with basic principles It is possible to approach emergency situations with confidence and with the expectation that rational diagnostic and therapeutic procedures can be effectively applied if the fundamental

principles to be outlined here are followed Acute emergency circumstances do not often permit, and usually do not require precise diagnostic and therapeutic efforts but they do demand an approach based upon an attitude arising out of awareness of the fundamental principles

Limitations Imposed on Physicians—One of the most elemental of these principles is an awareness by the physician of the severe limitations he encounters in his endeavors to ascertain the pathologic processes at work in the patient with which he is confronted This is particularly true in an emergency situation The temptation is great to arrive at a positive diagnosis early and to place in this snap judgment an unmitigated and persistent confidence Such an attitude effectually shuts the door against other plausible diagnostic and therapeutic inquiries and makes it difficult to shed an original diagnosis even in the face of increasing contrary evidence

A working or provisional diagnosis which is recognized at the outset as just that is useful in the therapy of any patient and especially the emergency patient However, this early diagnosis should usually be limited to an interpretation of disorders of basic functions of respiration and circulation in emergency states and a minimal effort made to arrive at precise identification of a disease entity or a classic syndrome Agnosticism or open mindedness in relation to a working diagnosis with an appreciation of the need for change in diagnosis as evidence for it accumulates in the patient need not paralyze one in instituting prompt

and intelligent therapy on behalf of the patient On the contrary such an attitude leads to the implementation of a sequence of procedures both diagnostic and therapeutic, which regularly leads to fruitful results

Changes in Patient—Another principle emphasizes the need for an awareness that from the moment of injury and throughout his clinical course the patient is never the same Despite the fact that he carries the same name and often the same diagnosis as originally assigned he is a perceptibly different individual 10 minutes after the onset of the emergency state than he was five minutes earlier or will be 30 minutes later Although the factor or the event precipitating the acute situation may be static it sets in motion a dynamic series of changes As a consequence it becomes imperative to observe and treat the patient as the dynamic continuously changing organism that he is Unless the physician is on the alert for changes subtle but significant modifications may take place but go unnoticed

Multiplicity of Lesions—A third principle useful in the evaluation of the patient in an emergency state is the realization that multiple lesions are present more often than are single lesions It is of little benefit to the diabetic patient in coma to be treated exclusively as a diabetic if the coma is due not to the diabetes but to a ruptured aneurysm of the circle of Willis Too many patients have died in acute circumstances because treatment was instituted and maintained for cerebral concussion simply because the signs and symptoms of concussion were similar to the signs

of a ruptured middle meningeal artery The pure lesion seems to be the exception rather than the rule in patients encountered in emergency situations

Syndromes' May Be Misleading —A fourth principle emphasizes the fact that signs and symptoms are manifestations of disturbances in physiologic mechanisms and are not necessarily pathognomonic of the pathologic character of the lesion or lesions which underly the disturbances Implicit reliance on syndromes *set forth in textbooks may mislead the physician* as often as it leads him to a proper diagnostic evaluation of his patient There is a tendency to carry collections of signs and symptoms about in one s head and to appeal to them when confronted with a disorder and a necessity for diagnosis Bedside experience and experimental evidence have abundantly shown that the demonstration of many and even all of the clinical changes that constitute a particular syndrome does not dependably signify the presence of the pathologic lesion with which the set of findings has been classically associated in the past Nor does the absence of certain clinical signs singly or in combination permit the conclusion that the commonly associated pathologic lesion is not present Syndromes are derived from the average changes encountered in pure lesions As such they have to do with statistical probabilities rather than certainties The reposing of confidence in clinical syndromes entails genuine risk for both patient and physician

If the physician in an emergency situation keeps in mind and applies the principles outlined it may be

expected that he can proceed rationally and effectively The establishment of a valid diagnosis is desirable but his primary responsibility is appropriate therapy In emergencies precise diagnosis may not be attainable but effective therapy based upon an understanding of basic physiologic mechanisms can often be fashioned without a specific label for the disorder

1

Airway and Artificial Respiration

ONE of the serious indictments of modern specialized medicine is the lack of knowledge of many doctors of how to perform effective artificial respiration in emergency situations For example is it not true that many times the doctor is embarrassed in the presence of a crowd around a drowned or electrocuted person because he realizes that he is less competent to per form a life saving act than the Boy or Girl Scout the policeman or the fireman who stands watching his fumbling efforts at resuscitation? In these respects as in all matters of restoration to normal function, the physician should be the best informed and it is the intent of this chapter to set forth certain simple yet effective procedures that are applicable in emer gencies requiring artificial respiration Reestablish ment of functional pulmonary ventilation is an abso lute must and a first whenever normal respiratory activity is altered or ended Diagnosis of the cause of the disruption of respiratory activity and defini

tive treatment can always wait until reoxygenation of vital centers is accomplished by artificial respiration Diagnosis and therapy are of little avail in the absence of the oxygen supply to the tissues necessary to the maintenance of life

ESTABLISHMENT OF AIRWAY

The first requisite to the performance of efficient artificial respiration is the establishment of a free airway In all patients requiring assistance to respiration whether on the beach at the roadside or in the operating room one must determine that it will be possible to move air into and out of the lungs without obstruction to the flow of that air First of all one must recognize that a free and unobstructed airway does not exist until with movement of air into and out of the lungs there is unrestricted motion of the chest cage particularly in the upper intercostal area and no noise associated with movement of the air except the very soft sound characteristic of movement of a mass of air through a fairly large orifice

Sites of Obstruction

Oropharynx —In any unconscious anesthetized asphyxiated or otherwise depressed patient the most common site of obstruction to air flow is the hypopharynx in that relatively shallow area between the

base of the tongue and the posterior pharyngeal wall Relaxation of the jaw and tongue permits the tongue to fall back and occlude that space and interfere with the passage of air to and from the lungs This type of obstruction is commonly known as swallowing the tongue

Relief from this sort of obstruction can be accomplished by several means In an emergency situation substantial relief often can be given by placing the patient in either the lateral or the prone position with the head to the side so that the tongue and jaw fall forward and away from the posterior pharyngeal wall thus creating a passageway for air With the patient in the supine position the head should be extended and turned to either side and the jaw supported at the angles by forward pressure with the fingers (Fig 1 *A*) The worst possible position is supine with the head flexed and on a pillow *Avoid the use of pillows or anything else under the patient s head unless the* shoulders are also elevated at the same time and to the same extent (Fig 1 *B*) This is essential to success of the maneuver

The lateral and prone positions are much to be preferred not only because the airway is more easily *established and maintained* but also because they facilitate drainage of any fluid food or other material contaminating the respiratory tract If placing the patient in the optimal position does not provide a free airway and forward pressure from behind the angles of the jaws is not sufficient the following measures *may be tried*



FIG 1—*A* pressure by the forefingers behind the angles of the mandible will force the jaw forward and help relieve oro-pharyngeal obstruction *B* the patient's head in optimal position turned to the side and extended A pillow is used placed well under the shoulders as well as under the head

Grasp the tongue with fingers or forceps and pull it forward This effectively relieves obstruction of this order but is a traumatic and usually unnecessary procedure Equally effective and more permanent relief of obstruction by the tongue is afforded by use of

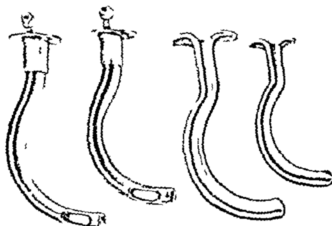


Fig 2 —Oropharyngeal airways two with cannula through which aspiration or insufflation can be accomplished Rubber bumpers should be placed near the flange end of metal airways to protect the teeth

a pharyngeal airway These devices (Fig 2) sometimes called mouth tubes are designed to fit over the tongue and back into the hypopharynx where they keep the base of the tongue away from the posterior pharyngeal wall They can be left in place for relatively long periods and although they are no

guarantee to patency of the airway in most instances they are sufficient in themselves to maintain a respectable airway No physician's bag should be without two or three pharyngeal airways The size that will fit most adults is no 5 no 3 is a convenient size for children, and no 1 is suitable for infants

In some instances of acute obstruction to the airway by the tongue the masseter muscles are spastic and it is difficult to place a pharyngeal airway It is a common but brutal practice in these circumstances to place wedges between the teeth This is seldom necessary because the airway can be established by passing a well lubricated large bore rubber catheter through a nostril into the hypopharynx The catheter should be at least a size 34Fr with a large lumen and relatively thin wall A tube about 25 cm (10 in) long is sufficient for most adults and an abbreviated endotracheal catheter (Fig 3) is ideal for this purpose One should be careful not to pass the tube too far as it may enter the esophagus and precipitate regurgitation of gastric contents The tube need be passed only as far as is needed to establish a flow of air and the distance can be assumed to be about 3–4 cm (1¼ in) beyond the distance from the side of the nose to the tragus of the ear In children a smaller tube is used but children around 5 years old will tolerate and need a tube of size 20–24Fr After this tube is passed and some of the asphyxia relieved the jaw usually relaxes and permits more adequate control of the airway

Many spastic jaws can be opened without the

mutilation caused by screws and wedges by passing the forefinger of either hand down along the outside of the teeth inside the buccal surface until the space behind the last tooth is reached The tip of the finger is inserted in this space and outward rotation is effected

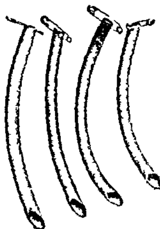


Fig 3 —Abbreviated endotracheal catheters useful for establishing an airway when it is impossible or difficult to open the patient's mouth They are inserted through the nares into the oropharynx behind the base of the tongue The safety pins serve as a point of fixation

(Fig 4) The leverage is sufficient to open the jaw enough to get a pharyngeal airway in place Pushing on the midpoint of the mandible is a totally ineffective means of opening a spastic jaw

Larynx —By the time most patients require artificial ventilation obstruction at the larynx due to

laryngospasm (spastic adduction of the vocal cords) is no longer apparent owing to antemortem relaxation However particularly in the operating room laryngospasm is a potent cause of respiratory obstruction It may be precipitated reflexly by dilation of



FIG 4—Method of inserting an oropharyngeal airway The forefinger of the left hand is placed between the buccal surface and the alveolar ridge behind the last tooth and gently rotated The airway is inserted with a rotating motion over the tongue

the rectum stripping of periosteum etc but it is more commonly incited by contamination of the upper respiratory tract with blood pus vomitus and other foreign material If after the patient is placed in the proper position and the airway has been otherwise controlled as outlined previously efforts at artificial respiration are still impeded by obstruction it is well to investigate the possibility of obstruction at the larynx This can be done by digital examination to determine if

any foreign objects (food particles teeth, etc) are impinged in the glottis but it is better accomplished by direct inspection with a laryngoscope A convenient laryngoscope for this purpose is that used by the anesthetist (Fig 5) The well prepared physician will

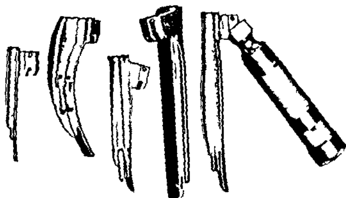


Fig 5 —Different types of laryngoscope blades with detachable handle enclosed batteries

have obtained instruction in the use of this instrument for emergency purposes from either the endoscopist or the anesthetist and will carry one at all times in his bag After the glottic area and larynx are examined and foreign material is removed it is often expedient to pass an endotracheal catheter which will guarantee an airway for artificial respiration and decontamination of the tracheobronchial tree as needed The relatively simple endoscopic maneuvers necessary for the performance of these emergency measures can be learned in a short time from competent instructors, and every

physician who is likely to encounter such problems is not well prepared to meet them until he is so equipped and trained

TRACHEOTOMY

There are many situations particularly of an emergency nature in which tracheotomy is the optimal method of providing an airway The measures already outlined are sufficient to control the airway in most instances but *there should be no protracted attempts* to use these methods in preference to tracheotomy Much valuable time is lost and many patients die as a result of persistent unsuccessful attempts to provide an airway with oropharyngeal airways naso pharyngeal catheters and endotracheal tubes Tracheotomy should be considered early in all instances of respiratory obstruction and accomplished without delay after it becomes apparent with a few attempts that conservative measures will not be successful

Tracheotomy in the acute emergency a situation with which this book is chiefly concerned can be done by the insertion of a knife blade through the ligamentous structure between the cricoid and the thyroid cartilage This procedure is recommended in many texts and perhaps is justified in an absolute emergency However this approach is often followed by atresia of the larynx. An equally effective approach can be used in the acute emergency without the high incidence of undesirable sequelae

TECHNIC The patient s head is so placed that there is acute hyperextension of the neck This may be done by placing the neck across a knee Tracheal rings are then easily palpated in the suprasternal notch and an incision is made in a longitudinal and cephalad direction from the sternum for about 7 5 cm (3 in) The tissues in this area which is essentially avascular are rapidly separated to expose the third fourth and fifth tracheal rings which are then incised under direct vision The tracheotomy tube is placed in the trachea also under direct vision An attempt to place a tracheotomy tube blindly is usually unsuc cessful and may actually result in perforation of the posterior wall of the trachea and the esophagus

Recently a device known as the Sierra Shelden Tra cheotome (Fig 6 *Top* and *Bottom*) has been in troduced for use in emergency circumstances It is an effective instrument for adults but less desirable for small children and infants

It is in circumstances of this sort that the physician will find it most beneficial to have a source of suction immediately available (see the following section)

Tracheotomy tubes come in different sizes and the physician should have in his emergency bag a size 0 for infants sizes 1-3 for small children sizes 2 4 for adolescents and sizes 4 6 for adults These tubes should be the full curve type with the inner tube extending to the tip of the outer tube Tubes that are not full curved and do not have the inner tube extend ing to the tip of the outer tube are harder to insert more easily occluded and much harder to clean

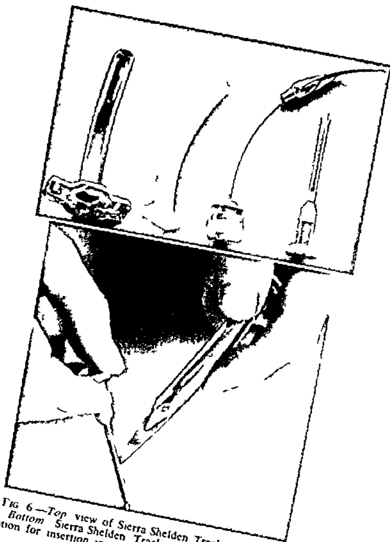


FIG 6—*Top* view of Sierra Shelden Tracheotome apparatus *Bottom* Sierra Shelden Tracheotome assembled and in position for insertion into trachea

In less acute emergencies the tracheotomy can be performed under sterile conditions and the opening made by surgical dissection of the overlying skin and subcutaneous structures An elective tracheotomy of this sort is placed in the neck at a point approximately 2 fingerbreadths above the sternum in the suprasternal notch The procedure can be done rapidly and easily with local infiltration anesthesia It is facilitated if either a bronchoscope of an endotracheal catheter is in place at the time of the tracheotomy This insures a patent airway during the procedure and guards against accidental incision of the posterior wall of the trachea The incision in the trachea can be made down on the brochoscope or catheter the broncho scope or catheter retracted and the tracheotomy tube placed with care under direct vision and appropriately fastened to the neck of the patient

Persons attending the patient with a tracheotomy and the patient himself should be instructed in his care An important feature of this care is the fre quently repeated aspiration of the trachea The inner tube of the tracheotomy device should be cleaned at least every 12 hours and oftener if the secretions are copious and sticky

When the cause of the respiratory obstruction is relieved the tracheotomy tube may be removed and the tracheal opening allowed to heal A practical method of determining the time for removal of the tube is to cork the tube opening for variable periods to ascertain the ability of the patient to respire through his larynx If the patient can tolerate corking for 48

hours the tracheotomy tube may be removed with reasonable assurance that a patent airway exists

Suction

An invaluable and often neglected aid to the es tablishment and maintenance of an airway and par ticularly in emergency situations is suction The need for removal of foreign material from the upper and lower air passages always must be anticipated and ready dependable suction is the most efficacious meth od of doing this In his emergency bag the doctor will find it useful to carry a large (50 cc) syringe with which an appreciable amount of aspiration can be accomplished It is also advisable to carry one of the aspirating appliances that can be attached to the ordinary water faucet In homes without electricity this device provides reasonably adequate suction Although to many physicians it would seem unneces sary nothing is to be lost—and the physician who has encountered a situation demanding suction would en dorse this—by carrying a small portable electrically powered suction appliance in the car at all times The physician will need to carry in his emergency bag 10 15 ft of fairly rigid rubber tubing to attach to the suction appliances He should also carry several con necting adapters preferably of metal to avoid break age at crucial moments

The aspirating appliances to be used with suction are a metal tip and an ordinary urethral catheter The

metal tip is useful only for aspiration of material in the pharynx and is introduced through the mouth It must be used with caution to avoid trauma to the oral and pharyngeal mucous membrane and the teeth In patients whose jaws are tightly closed the metal tip can be introduced into the oropharynx by passing the tip inside the cheek but outside the teeth until it enters the oropharynx through the space behind the last molar

The urethral catheter that is used for aspirating purposes should be at least size 14Fr and preferably size 18Fr The rounded end should be cut off and the edges of the opening smoothed Aspiration is facilitated if an extra hole or two is cut into the side of the catheter in its terminal inch (2 5 cm) Too many holes cut at this point will weaken the catheter make insertion difficult and may cause the end to break off and be aspirated into the lung The catheter may be introduced through the mouth but aspiration is usually ineffective It is preferably passed through the nares into the naso and oropharynx In some instances it is possible to continue its passage down through the larynx into the trachea and large bronchi without direct visualization The catheter should be well lubricated with liquid petrolatum to minimize trauma to the mucous surfaces and facilitate its passage It is wise to interrupt suction while the catheter is being introduced and to aspirate only on withdrawal Suction by catheter inserted through the nose is more effective less traumatic and more easily instituted than suction by means of the metal tip in the oro

pharynx Both the metal tip and the catheter should be frequently cleared by being dipped briefly into water because they are easily occluded by mucus vomitus or other material that may be aspirated

ARTIFICIAL RESPIRATION

After the establishment of the airway—an absolute necessity in every situation in which improvement in pulmonary ventilation is desired—one can consider the optimal means of effecting respiratory exchange The primary object in artificial respiration is to supply oxygen to the alveoli and the arterial blood and also to provide for elimination of carbon dioxide What ever may have been the cause of the respiratory arrest or deficiency this undesirable state will be perpetuated by oxygen lack to the cells concerned with the integration of all the afferent impulses (mechanical thermal chemical etc) and the ultimate production of a respiratory effort

Carbon dioxide—One is always confronted at this stage of a discussion of artificial respiration with the controversial issue of carbon dioxide It is well established that carbon dioxide is a potent respiratory stimulant and that it is capable of stimulating respiration in hypoxia These observations have led to the general practice of including carbon dioxide in the inhaled mixtures during artificial respiration In all states of depressed or absent respiration there is accumulation of carbon dioxide in the circulating blood

from metabolic processes in the body This amount of carbon dioxide is more than sufficient to stimulate respiration *if* the cells of the various respiratory centers are sufficiently oxygenated to function As indicated before the important element that is needed is oxygen, and in the presence of an adequate supply of oxygen there is little or no need for addition of carbon dioxide It has been stated that carbon dioxide is needed to facilitate the dissociation of hemoglobin in states of hypoxia This artificial device is also not needed if the blood stream is provided with a high oxygen tension Evidence has been accumulating that a sudden reduction of an elevated carbon dioxide tension in the plasma may precipitate serious cardiac irregularities and pronounced hypotension Most of this evidence has been accumulated in laboratory animals but if the observations are confirmed in human beings with respect to situations requiring artificial ventilation it may be necessary to revise concepts regarding the role of carbon dioxide in resuscitation

Analeptics —Analeptics such as nikethamide (Coramine®) pentylene tetrazol (Metrazol®) alpha lobeline and caffeine probably have no place in the restoration of respiration They are of little or no use if respiration has ceased and are significantly less effective if oxygenation is inadequate and valuable time may be lost if they are given priority over establishment of pulmonary ventilation by the methods to be outlined on the following pages

Satisfactory antagonists to respiratory depression induced by narcotics have been developed These antag

onists are n allylnormorphine (nalorphine hydrochloride, Nalline®) and levallorphan tartrate (Lorfan®) These drugs given intravenously in appropriate doses effectively counteract respiratory depression and there is evidence that they may be effective in overcoming other effects of narcotics n Allylnormorphine may be given in an initial dose of 0 005 Gm, and the dose may be repeated once Levallorphan tartrate may be given in an initial dose of 0 001 Gm which may be repeated once The drugs should be given relatively slowly to avoid hypotension and there is evidence that doses higher than these maximums may contribute to depression These drugs are capable of providing analgesia and respiratory depression and should be used with caution When used in the absence of narcotics they act as depressants in the same manner as do the narcotics Their use as antagonists should be restricted to those situations in which depression is caused by narcotics They may be used alone as analgetics The physician should not expect that the patient may be aroused from a nonresponsive state even though the respiratory rate is returned to normal or near normal The return to effective ventilation may be expected within 2 to 3 minutes After satisfactory ventilation has been established there may be regression and repeated use of the antagonists may be necessary

The minimal volume of gas to be moved in order to provide adequate gas exchange must exceed the physiologic dead space of the respiratory passages Anatomic dead space is the volume of the respiratory passages extending from the nostrils to and including

the terminal bronchioles It is the space in which no gas exchange takes place except very slowly by dif fusion This space varies somewhat, but in most normal male adults with a tidal volume of 600 cc or more and a respiratory rate of 15 16 per minute the mean dead space volume is about 150 cc Physiologic dead space includes anatomic dead space plus any area in proximal radicals or alveoli in which active exchange does not occur To this during resuscitation are added varying volumes of dead space One can see that to move the respired atmosphere to a point in the lung at which exchange can take place between alveolar atmosphere and blood (alveolar ventilation) and to a point in the external lung (resuscitative apparatus) at which carbon dioxide may be removed and oxygen and anesthetic added the volume must exceed the dead air space Ventilation should be measured both in terms of tidal exchange and minute volume exchange For example slow deep respiration may produce satisfactory alveolar ventilation whereas rapid shallow respiration may produce insufficient alveolar ventilation

Methods for Artificial Respiration

There are a number of methods for artificial respira tion in an emergency situation that can be performed as well by the doctor as by the fireman or Boy Scout These are divided roughly into two methods of getting air into and out of the lungs One set which includes the Schafer Silvester and Eve rocking methods pro

duces the movement of air in the lungs by extrathoracic influences on the thoracic cage The other set of methods which is definitely more efficient and includes mouth to mouth insufflation or use of various inhalators and resuscitators produces ventilation of the lungs by forcing air through the upper respiratory tract into the lung at intervals Some of these devices also provide for active deflation of the lung

In considering the material to follow which relates to the efficiency of the manual methods, one should remember that the tidal volumes reported were attained under ideal conditions These conditions included an airway established by an endotracheal catheter a situation not often prevailing in emergency circumstances Drs James O Elam of the Roswell Park Memorial Institute and Peter Safar of the Baltimore City Hospital have recently examined the efficacy of these methods (data not yet published) under conditions more closely approximating those encountered on the beach at the roadside etc With both trained and untrained personnel being used with and without an oropharyngeal airway but always *without* an endotracheal catheter the tidal volumes moved were except for occasional instances well under the amount necessary to ventilate the patient satisfactorily The tidal volumes reported in Figure 7 (page 38) are not those one might expect from manual methods in the absence of an endotracheal catheter and not those one might expect from manual methods applied in the usual emergency conditions

These observations of Elam and Safar emphasize

the extreme importance of establishing and maintaining an airway in circumstances in which ventilation is diminished or absent Elam and Safar propose, as they have consistently in the past that the most efficient method of artificial ventilation (in the absence of mechanical equipment) is mouth to mouth More detailed description of this technic will be given later (p 50)

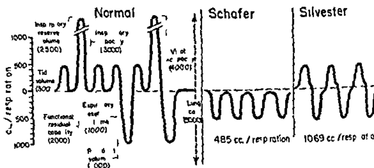


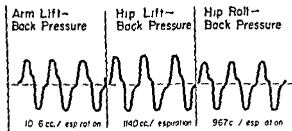
Fig 7—Comparison of tidal volumes with various manual methods of artificial respiration Values noted were obtained in series on curarized anesthetized intubated normal men

The manual methods may be divided into three basic groups (1) push methods that provide active expiration with inspiration passive (2) pull methods providing active inspiration and passive expiration and (3) combinations in which there is a push pull mechanism providing both active inspiration and expiration In general the push pull technics are 2 to 3 times as efficient from the standpoint of pulmonary

ventilation as either of the other technics individually

Each of the push pull methods has 4 phases The lift or roll provides active inspiration the release permits passive expiration the press provides active expiration and the subsequent release permits passive inspiration

The optimal rate for performance of these manual methods of ventilation appears to be 10 12 times a



(Figs 6 12 from Gordon A S *et al* J A M A 147 1444 Dec 8 1951)

minute with a 5 6 second cycle and with each of the 4 phases lasting approximately 1½ seconds There is merit in increasing the rate above this level in the early stages of resuscitation to insure adequate ventilation Hyperventilation is difficult to produce by these technics and need not be a concern of the physician Furthermore the energy output of the operator is a self limiting factor in the rate of resuscitative efforts

Studies of the energy output for the various man ual technics showed that the Schafer prone pressure method was least exhausting whereas hip roll and hip lift (alone) required the most energy The Silvester and arm lift technics were intermediary in their en ergy requirements

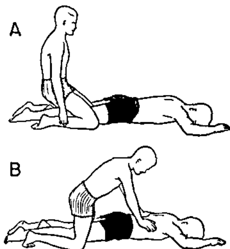


FIG 8—Schafer prone pressure method of manual artifi cial respiration *A* inspiration *B* expiration

It has also been determined that the methods easi est to learn were the arm lift back pressure and arm lift chest pressure methods The hip lift method was most difficult to learn

SCHAFER METHOD (PRONE PRESSURE) (PUSH) In this method the patient is prone with

the head extended and turned to the side and resting on one hand It is evident from the previous discussion on the airway why this position has merit The operator faces the head and kneels and straddles one or both legs of the patient His hands are placed over the floating ribs with the fingers together and directed laterally Steady pressure is exerted downward and toward the patient s head for approximately 2 seconds after which the pressure is released After a pause of about 3 seconds the procedure is repeated Ventilation is accomplished by the pressure of the viscera against the diaphragm resulting in active expiration after which passive inspiration is accomplished by the elastic recoil of the compressed lower chest cage (See Fig 8)

SILVESTER METHOD (ARM LIFT CHEST PRESSURE) (PUSH-PULL) This method has the advantage of being adaptable to situations in which for one reason or another it is impossible or inadvisable to place the patient in the prone position It has the significant disadvantage that with the patient supine the upper airway is more likely to become obstructed from the tongue and jaw relaxation In addition the supine position does not facilitate the drainage of secretions foreign material and the like from the upper and lower respiratory tracts

The patient is placed in the supine position with the arms folded on the chest The operator kneels at the patient s head and faces his feet He grasps the patient s arms just above the wrists lifts them up-

ward then above the head and finally downward until they touch the surface on which the patient is lying The patient s arms are then placed on his chest and pressure is exerted downward The first maneuver produces active inspiration and the second one active

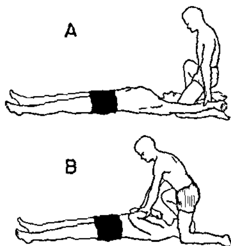


FIG 9—Silvester method (arm lift-chest pressure) of artificial respiration *A* inspiration *B* expiration

expiration As in the Schafer method there should be pauses between the inspiratory and expiratory phases (See Fig 9)

NIELSEN METHOD (ARM LIFT BACK PRESSURE) (PUSHPULL) In this technic the patient is prone with the head extended and placed on the hands The operator faces the feet of the pa-

tient kneels upon either knee and places his hands under the arms just above the elbows The patient s arms are then lifted upward and the operator rocks backward drawing the arms toward him until he meets resistance The patient s arms are then placed on the surface on which the patient rests and the operator moves his hands to the patient s back at a point just below the scapulae He then rocks forward exerting pressure downward As in the Silvester method the first maneuver produces active inspira tion and the second active expiration Cycling is the same as for the other technics (See Figs 10 and 11 *A*)

HIP LIFT (PULL) In this technic the pa tient is in the prone position as in the Schafer method The operator faces the head of the patient kneels on one knee at the level of the patient s hip and places the other foot near the opposite hip The operator s hands are placed under the hips of the pa tient at the anterior superior iliac spines and the pelvis is raised 4 6 in The hips are then lowered to the surface on which the patient is resting and the cycle is repeated at approximately the same rate as for the other technics This technic produces active inspira-tion only

HIP LIFT BACK PRESSURE (PUSH PULL) The patient is placed in the same position as for the hip lift technic and the procedure for lifting the hips is repeated However alternating with the lifting of the patient s hips pressure is applied by the operator

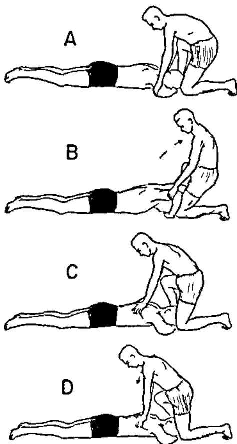
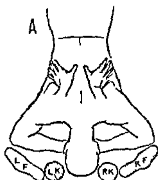


Fig 10—Arm lift back pressure method of artificial respiration (after Nielsen) *A* placing hands for arm lift *B* arm lift *C* placing hands for back pressure *D* back pressure



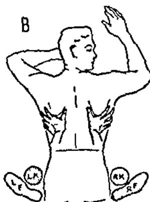


FIG 11—*A* placement of hands and feet for arm lift back pressure method *B* placement of hands and feet for hip lift back pressure method *LK* left knee *RK* right knee *LF* left foot *RF* right foot.

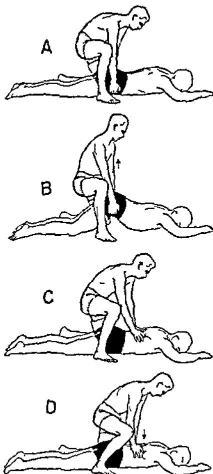


FIG 12—Hip lift back pressure method of artificial respiration *A* placing hands for hip lift *B* hip lift *C* placing hands for back pressure *D* back pressure

with his hands just below the scapulae The fingers are spread and the thumbs are opposite each other about 1 in from the midline As the operator lifts the hips he rocks backward, and as he applies pressure he rocks forward His arms are kept straight at all times Cycling rate is as for the other technics This method provides active inspiration with the hip-lift and active expiration with the back pressure (See Fig 12, see also Fig 11 *B*)

HIP ROLL—BACK PRESSURE (PUSH PULL) In this technic the patient and operator are in the same positions as for the hip-lift and hip lift back pressure technics The latter method is modified in that instead of lifting the hips the operator uses the knee on which he is resting as a fulcrum on which to roll the hips of the patient The operator s arms are kept straight and he rolls in the same direction as he is rolling the patient Attention must be paid to rolling the patient sufficiently to elevate both hips from the surface on which he is resting Back pressure is applied alternately with the roll of the hips of the patient This method provides active inspiration and active expiration (See Fig 13)

EVE S METHOD A good method of manual artificial respiration which has not been very popular in this country but widely used in England is use of Eve s rocking stretcher In this method the patient is placed prone on a board which is then placed on a sawhorse or barrel or some other fulcrum of sufficient height to permit 45 degree tilting of the board (Fig

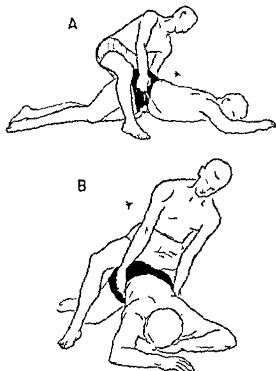


FIG 13 —Hip roll back pressure method of artificial res piration *A* side view of hip roll *B* front view of hip roll

14) The abdomen of the patient is located on the board over the spot where it is placed on the fulcrum The head is placed to insure an airway and the arms are fixed along the sides The board is tilted through a 90 degree arc with the patient s head alternately down and up The abdominal contents alternately press and

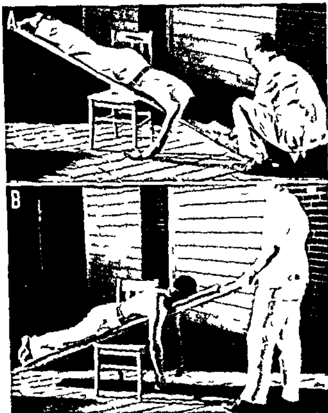


FIG 14.—Eve's rocking method of artificial respiration *A* the patient is placed on the board in the position described in the text and shown here His hands may be fastened to the side if desired to prevent injury during movement of the board The board is moved through the arc shown in *A* and *B*

release the diaphragm and effect ventilation in this man ner This method and position also facilitate control of the airway and elimination of foreign material in the respiratory tract With the patient in the prone position and the board tilted through a 90 degree arc tidal exchange is between 550 and 850 cc It is claimed that this method has another advantage in that it fa cilitates venous return to the heart and therefore im proves cardiac output

The remaining methods accomplish artificial ven tilation by providing positive inflation by the introduc tion of air or oxygen and sometimes mixtures of oxygen and carbon dioxide under increased pressure into the tracheobronchial tree These methods are consistent ly and significantly more effective than the manual methods just outlined However they are sometimes less readily available than the manual methods because with the exception of the mouth to mouth technic some type of apparatus and gas supply is required

MOUTH TO MOUTH METHOD This is one of the simplest and most efficient of the resuscitative methods Tidal exchange is adequate and it is always available In addition it has been demonstrated that higher than normal minute volume exchange can be easily effected with relatively minimal energy output Because of this elevation in minute volume exchange high oxygen saturation of the hemoglobin can be achieved in spite of a tension of oxygen in expired air which is lower than that of atmospheric air With suitable ventilation it has also been demonstrated that

there is no deleterious increase in carbon dioxide tension *

Elam and Safar have evidence to substantiate the effectiveness of mouth to mouth or mouth to airway artificial ventilation With the same personnel who performed the manual methods so inefficiently there was not a single failure to produce tidal volumes of 1,500 ml and over with the foregoing technic

The prominent disadvantage is esthetic and many are discouraged from employing the technic for that reason An equally effective and somewhat more esthetic approach is the interposition of an ordinary anesthetic face mask between the mouths of the patient and the operator The mask makes possible a better apposition and the added dead space augments the concentration of carbon dioxide † There is unjustified criticism of the technic on the basis of the possibility of overinflation and rupture of alveoli even in the infant This latter factor can be simply and readily controlled by watching the chest and limiting inflation pressures to that necessary only to begin elevation of the chest cage

In this circumstance the patient is supine As a result the resuscitator must take extra precautions to establish and maintain the airway as outlined earlier He must be especially alert to foreign material in the

Greene D G Bauer R O Janney C D and Elam J D Expired air resuscitation in paralyzed human subjects J Appl Physiol 11 313 318 September 1957

†Elam J O Brown E S and Elder J D Artificial respiration by mouth to-mouth mask method, New England J Med 250 749 754 May 6 1954

airway and remove it by the most suitable means at hand After establishing the airway the resuscitator kneels close to the patient s left ear and places his mouth over the patient s mouth or on the face mask He must occlude the patient s nostrils to diminish air leak through them This may be done by pinching the nose together or by leaning against the nares with the right cheek In infants and small children it is usually advisable to include both nose and mouth in the mouth of the resuscitator The resuscitator then blows gently and watches the chest of the patient As soon as the chest begins to elevate blowing is stopped and the mouth of the resuscitator is removed from that of the patient permitting the patient to exhale passively (See Figs 15 and 16) If with blowing into the patient s respiratory tract there is no elevation of the chest and there is rumbling in the stomach the resuscitator must recheck the patency of the airway improve the contact with the patient by eliminating leaks between mouths or between mask and mouths and/or use more forceful blowing (See Fig 17) Dilatation of the stomach may accompany this type of artificial ventilation and it will be detected by protuberance of the stomach wall and a hollow sound to percussion of the abdomen If dilatation is present pressure over the stomach should be applied to remove the excess gas in the stomach but one must be prepared to remove from the pharynx any other material that may come from the stomach with this maneuver If gas is not easily expelled by pressing upon the abdominal wall introduction of a gastric tube *may facilitate the procedure*



Fig 15 —Mouth to mouth technic
Fig 16 —Mouth to-mouth technic

Mouth to Airway Technic —Safar and McMahon found in the course of their observations on mouth to mouth methods that blowing into the upper airway of a patient through an oropharyngeal airway placed in the mouth of the patient was more acceptable to the operator and produced excellent ventilation and minimal gastric distention They therefore combined a No 4 oropharyngeal airway with a No 3 oropharyn-

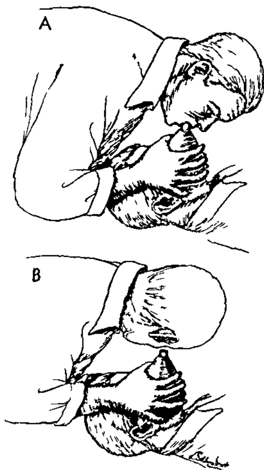


Fig 17 —Mouth to mask technic *A* inflation *B* operator inhales while patient exhales

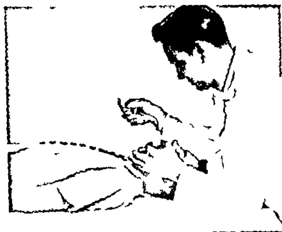




FIG 18—Mouth to-airway te hnic (Note placement of instrument and position of patient and resuscitator)

FIG 19—Mouth to-airway technic (Note elevation of chest with inflation maneuver and position of resuscitator's hands in maintaining airway and occluding nose)

geal airway by welding the two together at their respective bases (lip end) Each faced in an opposite direction to form an S shaped instrument (See Figs 18, 19 and 20 *A* and *B*) With the exception of infants these two airways will fit most patients who require emergency resuscitation

The appropriate sized airway of the pair is introduced as described earlier (Fig 4 page 25) After the airway has been properly inserted and the jaw supported to eliminate obstruction the resuscitator kneels at the head of the patient closes the nose with the thenar eminences of his thumbs and blows into the airway that remains outside As in the technic for mouth to mouth or mouth to mask ventilation the resuscitator must watch for elevation of the chest and cease blowing as soon as elevation is observed to permit exhalation to take place

This technic is simple produces effective ventilation is more esthetic can be learned readily by lay personnel and requires minimal energy output The apparatus is inexpensive and efficient

APPARATUS FOR ARTIFICIAL VENTILATION One of the simplest devices is the Kreiselman hand operated cylindrical bellows On one end is a face mask (it may also be attached to an endotracheal catheter) and on the other is a handle By alternately pushing and pulling one can apply positive and negative pressure to the airway and the lungs

All other devices used for artificial ventilation employ either the Venturi principle a piston or compression of a bellows A gas under pressure from a cylin

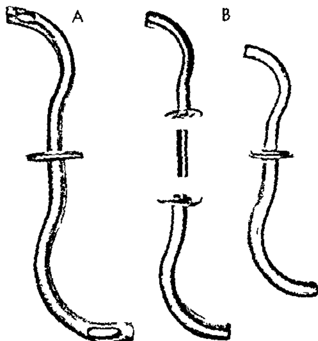


FIG 0—*A* Welded metal airways *B* Rubber airways with metal insertion

der (usually oxygen) is delivered to a Venturi device to a piston to a cam compressing a bellows or to a chamber in which there is a bellows or a bag

The gas under pressure is delivered through a patent airway to the lungs After a predetermined pressure has been reached the volume of gas is vented to the ambient atmosphere If venting is facilitated by

and a connection for a 3 L anesthetic rebreathing bag (Fig 21) By applying the face mask and filling the bag with oxygen one can accomplish active and



Fig 21—Simple but extremely effective method of artificial ventilation of the lung The mask is applied snugly to the face and intermittent positive pressure is applied by hand to the rebreathing bag full of oxygen The oxygen is delivered to a nipple connection on the elbow fitting between bag and mask

complete inflation of the lungs by manual compression of the rebreathing bag The degree of inflation can be accurately and positively controlled by observation of the patient s chest and the rate can be adjusted to suit the patient s needs The apparatus is simple and

inexpensive and provides an entirely efficient method of artificial respiration It eliminates the necessity for complicated expensive heavy respirators The bag and elbow can be connected through suitable connectors to an endotracheal catheter if desired With this apparatus or with the conventional resuscitators, tidal exchanges of 1 000 1 500 cc can be obtained

It must be remembered that although oxygen is the natural accompaniment of the resuscitators and the simple bag and mask oxygen can still be given by face mask or catheter to patients receiving artificial respiration by any of the other technics There is absolutely nothing to be lost and much to be gained by giving oxygen with all forms of artificial respiration The addition of oxygen to the respired atmosphere may mean the difference between successful and unsuccessful revivification

There has been a tendency in the past to limit the extent of tidal exchange during artificial ventilation by mercury of water manometers set at predetermined pressures Since the volume of air flow in a given interval required to ventilate a patient suitably may necessitate the use of pressures momentarily above the usual limits of 20 cm of water these devices have been largely abandoned If close attention is paid to the movement of the thoracic cage or to the mediastinum (if the thorax is open) there is little need to apply undue pressure The occasional patient may suffer the rupture of an emphysematous bleb but more patients will benefit by adequate ventilation

RESUSCITATION OF THE NEWBORN

There is a universal tendency to consider resuscitation of the newborn a special problem For this reason it is treated separately in this discussion of resuscitative measures Actually the primary distinguishing feature of the problem is the etiology of the depression of respiration in the newborn infant Prophylactic treatment is most effective and every effort should be taken to avoid depression of respiration from analgesics hypnotics and anesthetic agents administered to the mother Rapid labor accompanied by persistent increased uterine tone should be minimized if possible Operative deliveries should be expeditious and nontraumatic It is not within the scope of this book to elaborate on these factors

The fundamental principles outlined in the previous discussion apply to infants as well as to adults Establishment and maintenance of the airway and ventilation of the lungs with oxygen are essentials in both circumstances The baby who is severely asphyxiated is often in shock and the shock must be treated by maintaining warmth and avoiding excessive handling and too vigorous resuscitative efforts

As is the case with adults, the baby s lungs can be inflated by mouth to mouth insufflation by application of a small mask and connected bag of oxygen or by endotracheal intubation and subsequent ventilation with oxygen A number of devices for infant resuscitation are on the market all of them activated on the same

principle as are the resuscitators for adults Considerable justifiable concern is taken over the necessity for limiting the inflation pressures in the infant but again as with adults the inflation of the lungs can be simply, effectively and safely controlled by watching the chest cage The lungs need be inflated only to the point at which the chest wall begins to lift and there is really little necessity for elaborate mechanical devices for this function

The depressed and asphyxiated baby should be handled gently should not be hung up by the heels and should be kept in a warm environment with head slightly down Immersion in ice water and slapping are admittedly effective stimulating procedures and physiologically sound in that they produce potent neurogenic stimuli However they are effective usually only in the mildly or moderately depressed infant who is readily resuscitated by less strenuous means and are usually totally ineffective in the severely asphyxiated baby In the latter the immersion and other vigorous resuscitative efforts tend to enhance the shock which is present and may be detrimental to the resuscitative effort

The infant's airway should be cleared and kept clear before and during resuscitative efforts This can be accomplished by several means but the most effective is the insertion of a small urethral catheter into the pharynx and into the larynx by direct vision with a laryngoscope Aspiration is often effectively accomplished by placing a catheter in the pharynx and

applying suction from the mouth through an interposed glass receptacle A small rubber ear syringe is useful for aspirating mucus from the mouth of the infant

It is also possible to insert a catheter into the larynx of the infant by putting two fingers into the mouth elevating the epiglottis and passing the catheter between the fingers Extreme adeptness with this technic can be attained and the need for visualization of the glottic area with a laryngoscope can be obviated

Innumerable infants would not have presented resuscitative problems had they not with their first voluntary inspiration aspirated into the tracheobronchial tree a quantity of mucus etc from the pharynx The first act of the obstetrician on delivery of the youngster should be prompt and adequate removal of foreign material from the pharynx and upper respiratory passages

2

Oxygen Therapy

OXYGEN therapy is an essential element in the successful treatment of most emergency situations and an understanding of the practical factors involved in the recognition of oxygen want and in the methods of its administration is important It is pertinent to point out that oxygen can be made available to almost every patient even in what appear to be remote places and unusual situations Small cylinders of oxygen are transported easily and equipment for its administration in emergencies should be kept constantly available Figure 22 illustrates a small cylinder of oxygen and the minimal equipment consisting of a mask and rebreathing bag necessary for its administration It cannot be emphasized too strongly that the recognition of incipient hypoxia and the prompt institution of oxygen therapy are essential factors in achieving good results No longer should the administration of oxygen be delayed until cyanosis develops nor should the oxygen tank be wheeled in only as a last resort to impress the relatives and frighten the patient

SIGNS OF OXYGEN WANT

The most reliable sign of early oxygen want is the pulse rate As hypoxemia or hypoxia develops the pulse rate increases and as the hypoxemia or hypoxia is relieved by excess oxygen the pulse rate returns to its original level One can assume that the tachycardia is not due to oxygen want if there is no change in the pulse rate with administration of the proper con centration of oxygen The pulse rate can also be used as a guide to the proper time for discontin uance of oxygen therapy If oxy gen therapy is discontinued and the pulse rate rises the patient still requires excess oxy gen but if the pulse rate remains steady excess oxygen is no longer needed These alterations in pulse rate oc cur within a few minutes



FIG 22—Unit consisting of small cylinder of oxygen yoke tubing bag and mask that can be transported eas ily and used in isolated areas for re suscitation or for oxygen therapy

The character and rate of respiration may change very little until relatively acute oxygen want develops at which time the rate increases



TACHYCARDIA
1

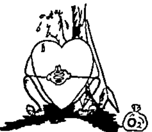


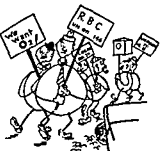
MENTAL CHANGES
(ANXIETY RESTLESSNESS DELIRIUM)
2



AIR HUNGER
3

FIG 23 —Signs of moderate oxygen want





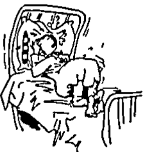


Fig 24 —Signs of acute oxygen want

Central nervous system changes are manifested in the early stages of oxygen want These are represented by sighing yawning and restlessness which progresses at times to acute delirium Restlessness is a common manifestation of oxygen want and too often is mistakenly treated with sedative drugs These respiratory depressant drugs only increase the oxygen want and fail to control the restlessness until dangerous doses are given In contrast oxygen will correct the true cause of the restlessness without resort to depressant drugs Restlessness and apprehension in any patient should suggest hypoxia and oxygen therapy should be tried before depressant drugs are given Precordial pain may develop in the early stage of oxygen want and some patients have fibrillary muscle twitchings

In acute oxygen want the pulse may become slow and bounding and the pupils fixed and dilated and the respirations which may have been rapid and shallow become depressed and irregular Cyanosis which may have been only slight may become severe and convulsions may replace the muscle twitching There is no question of the need for oxygen in this situation There should likewise be no question of the need for oxygen in the patient exhibiting the signs of early oxygen want Remember that cyanosis is a late sign of hypoxemia and that serious hypoxia may be present in the absence of cyanosis Cyanosis cannot develop in patients who have less than 6 Gm of hemoglobin per 100 cc of blood but these patients can have acute oxygen want

METHODS OF ADMINISTRATION

There are 4 common means of administering oxygen therapy (1) the tent (2) the mask, (3) the box and (4) the nasopharyngeal catheter The best source of supply of oxygen for all of these is the 244 cu ft cylinder of commercial oxygen or a central supply from which oxygen is piped to the bedside Medical oxygen is merely commercial oxygen dressed up in a smaller cylinder and is needlessly expensive Commercial oxygen may be obtained in any welding or blacksmith shop or direct from the distributor and consequently is available in nearly all communities A reducing valve is needed to reduce the high pressure of the large cylinder to a safe delivery pressure The reducing valve may be of the type used by the welder or it may be adapted for use in oxygen therapy by having incorporated in it a regulator valve with a flowmeter to show the flow of oxygen in liters per minute In an emergency the welder s reducing valve is acceptable the necessary rate of flow being estimated by the patient s response

OXYGEN TENT The principal advantage of the oxygen tent are the adaptability to children cooling of the atmosphere and the absence of appliances on the patient s face The disadvantages are its lack of availability in most emergency situations the relatively high initial cost the need for close and constant supervision to maintain proper concentrations and its interference with complete nursing care The last factor

is a particular disadvantage in an emergency It is essential that an oxygen tent be closely supervised to assure a satisfactory concentration of oxygen and a suitable atmosphere in the canopy Minimal leaks in the canopy and the improper tucking in of the tails of the canopy reduce the oxygen concentration out of proportion to the size of the leak (Fig 25) It is imperative that a rubber sheet be placed under the bed sheet in the canopy to prevent oxygen leak through the mattress Frequent sampling of the tent atmosphere should be done to guarantee adequate oxygen concentration Simple inexpensive apparatus are available for this purpose Tents should not be used for administering oxygen therapy unless adequate supervision is available

OXYGEN MASK The principal advantage of the mask for administration of oxygen therapy is the ease with which very high concentrations may be obtained It is also useful for patients who object to the nasopharyngeal catheter or tent Contrary to what might be expected the nasopharyngeal catheter is better tolerated than the mask for long-continued oxygen therapy The disadvantages are discomfort to the patient when worn over long periods and interference with nursing care particularly when the oronasal type of mask is used If the nasal mask alone is used the patient must co-operate by keeping his mouth closed (Fig 26) It is a simple method although somewhat more nursing care is required than with the nasopharyngeal catheter method because the mask must

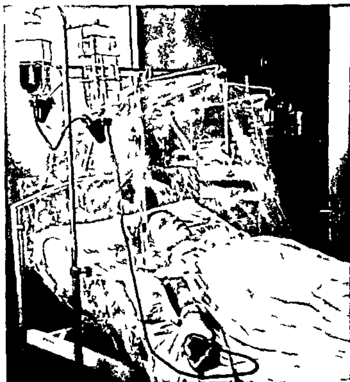


FIG 25 —Oxygen tent

be kept fitted tightly over the nose and mouth and frequent massage of the areas in contact with the mask is necessary to prevent pressure ischemia Practical use of this method is confined to patients who require high concentrations of oxygen and patients in emergency situations often require high concentrations

OXYGEN BOX The oxygen box is a convenient economical and simple means of supplying excess oxygen to infants and small children Relatively high concentrations can be obtained with reasonable flow rates Nursing care is unhampered Use of the oxygen box is not as wide pread as it might be A simple



FIG 26 —Oxygen mask

open top box can be made in mo t hospital shops A flow rate of 7 10 L per minute provides a con centration of 70 per cent or more It is essential however that the apron in the front of the box be secured tightly to prevent leaks

The oxygen box has been supplanted by a similar device known as the Croupette (Fig 27) This device

operates upon essentially the same principles as the old style oxygen box but it has a more efficient humidifying unit It does not have an open top It is especially use



Fig 27 —Croupette This unit is in some respects similar to the oxygen box It is useful for infants and small children

ful in those circumstances in which an atmosphere high in humidity and oxygen is desired

NASOPHARYNGEAL CATHETER The naso pharyngeal catheter method popularly but improperly termed the nasal catheter method is most readily available in all situations A concentration of 35-45 per cent of oxygen in the alveoli is obtained with a



Fig. 28.—Nasopharyngeal catheter method

moderate flow rate (7 8 L per minute) The technic is simple requiring minimal supervision (Fig 28) It is economical and there is no interference with nursing care

There are a few fundamental requirements for the proper use of the nasopharyngeal catheter method Failure to adhere to them results in inefficient therapy and accounts for many poor results obtained with the method The first requirement is proper humidification of the oxygen since the dry oxygen from the cylinder is irritating and causes discomfort In emergencies oxygen can be given for short periods without humidification A number of good humidifiers are on the market In an emergency a *humidifier* can be made quickly and inexpensively of two milk bottles or Mason jars some glass and rubber tubing corks and a rubber sponge for breaking up the oxygen into fine bubbles Good humidifiers should have a safety valve at the delivery outlet set at 40 mm Hg pressure With such a valve increased pressures cannot be built up and suddenly released in the patient s pharynx Also the delivery tube can be clamped off during swallowing to prevent the swallowing of large amounts of oxygen The valve also provides a check on the patency and cleanliness of the catheter because a dirty and plugged catheter will build up back pressure The valve also prevents severe gastric distention if the oxygen delivery tube is inadvertently connected to a gastric suction catheter

The second requirement is proper placement of the right type of catheter The catheter may be made

of latex gum rubber or plastic size 14Fr and should have a large lumen Several holes should be present along a distance of 1½ in from the tip An ordinary red rubber urethral catheter may be used but extra holes should be burned or punched in the area near the tip Proper placement of the catheter in the naso pharynx is a simple procedure With the oxygen flowing the catheter is introduced along the floor of the nose into the nasopharynx a distance equal to that from the side of the nose to the tragus of the ear Another method is to introduce the catheter until the patient begins to swallow oxygen The catheter then is retracted to a point just short of the swallowing reflex It is unnecessary to visualize the tip of the catheter in the nasopharynx With the catheter in position a flow of 7 L per minute will provide a concentration of 35 45 per cent in the alveoli Any deviation from this position will reduce the effective concentration

The disadvantages of the catheter method are few being chiefly the inability to obtain high concentrations when needed and occasionally an objection by the patient to presence of the catheter Most objections by patients can be traced to use of improper catheters to catheters which have become dirty to inadequate humidification of the oxygen and to in correct placement of the catheter The nasopharyngeal catheter method provides an efficient means for the routine treatment of oxygen want

3

Circulatory Emergencies

SUDDEN CARDIAC ARREST

THE CATASTROPHE of sudden cardiac arrest usu ally in the operating room is an emergency which confronts almost every physician at least once during his career Sudden circulatory arrest may be due either to cardiac standstill or to ventricular fibrillation The former condition occurs more frequently than the latter Cardiac insufficiency of this degree is usually detected by loss of palpable pulse loss of detectable blood pressure lack of bleeding in the wound extreme pallor with a gray tinge mottling and coldness of the skin The series of events which precipitates abrupt circulatory arrest is complex and open to speculation Effective treatment is available and prompt action may prevent an otherwise certain fatality

TREATMENT The most essential single thera peutic measure in sudden circulatory arrest is imme diate restoration of the circulation The administra tion of oxygen in high concentration with artificial

pulmonary ventilation by the measures outlined in Chapter 1 is of paramount importance to maintain adequate oxygenation of the lungs and also to aid in circulation of the blood In cases of sudden cardiac arrest or fibrillation effective cardiac compression must be instituted at once If the ventricles are fibrillating defibrillation must be accomplished It is emphasized that diagnosis of asystole or fibrillation can be made only by direct inspection of the heart through the open chest or by an electrocardiogram Several methods of interrupting ventricular fibrillation are available (1) electric shock (2) injection of procaine amide hydrochloride (Pronestyl®) into the blood stream or auricle (3) intravenous or intraventricular injection of quinidine gluconate and (4) intracardial injection of potassium chloride They may be used separately or together Electric shock can be applied by means of the bared ends of two wires leading from an ordinary 110 volt outlet There are now available several devices constructed with two flat electrodes to be placed on opposite sides of the heart Current from this apparatus is usually 110 volts with 0 5 1 5 amperes The shock is usually automatically limited in duration although it is initiated manually If quinidine is used quinidine gluconate (0 8 Gm per 10 ml ampule Lilly) is available Ordinarily it is not given more rapidly than 40 mg per minute The ampules may be used as packaged or diluted to 20 ml with isotonic sodium chloride solution Procaine 5 cc of 2 per cent solution may be given intravenously or

intra auricularly Procaine amide hydrochloride (Pro nestyl®) is ordinarily not given faster than 200 mg per minute and a total dose of 1 0 Gm is seldom exceeded without a short rest period Potassium chloride may be introduced into the aorta after it has been clamped distally A 3 per cent solution of potassium chloride (40 meq /20 ml) is used in children Twice this dose is used in adults The heart is compressed with the aorta clamped to circulate the potassium into the coronary system

Epinephrine is reserved for treatment of cardiac standstill and should not be used for defibrillation because it makes a hyperirritable myocardium even more irritable Extensive laboratory investigations in dogs have shown that if epinephrine is to be used in attempts to restore cardiac action it should be given into an auricle The drug should not be given into the ventricular muscle because of the greater tend ency to production of fibrillation The usual ampule of 1 ml of 1 1 000 epinephrine should be diluted 10 times and only 1 ml of this low concentration should be given at one time It is recommended that epi nephrine never be given blindly through the closed chest wall not only because the drug may be injected into the ventricular muscle but more significantly, be cause it is definitely contraindicated in fibrillation

The heart has an inherent capacity to function as a pump but when sudden circulatory arrest occurs no one can foretell whether a few stimulatory manip ulations of the heart through the diaphragm will in duce an effective rhythm or whether a prolonged

period of artificial circulation by *cardiac compression* will be necessary Time is at a premium and must not be squandered The deadline for complete recovery is approximately 3 minutes

If the thorax is open when the heart stops compression can be quickly and effectively executed If the abdomen is open when the heart stops and compression through the diaphragm does not produce a palpable radial or carotid pulse the compression is probably not effective in propelling the circulation and the heart should be securely grasped by actually entering the chest without delay through a new transthoracic incision When neither cavity is open a direct anterior intercostal approach through the left fourth or fifth interspace should be made This will allow the index and middle fingers of the left hand to be placed directly against the heart and so permit intermittent compression of the ventricle against the sternum The rate and force of cardiac compression should be sufficient to produce a palpable pulse and a measurable blood pressure

It is usually necessary to open the pericardium in order to accomplish effective cardiac compression

There are instruments which are said to be effective in initiating and maintaining cardiac contraction by the application of interrupted electric current to the chest wall The effectiveness of these devices is open to serious question Considerable danger is associated with their use in that attempts at resuscitation by means of these devices may seriously delay direct inspection diagnosis and adequate therapy

Epinephrine should be used with extreme caution during recovery following restoration of cardiac action to strengthen contraction of the myocardium If hem orrhage and shock are complicating factors blood should be given to assure adequate cardiac filling This may be accomplished by intravenous or intra arterial infusion

When co ordinated cardiac action has been re sumed the damage of anoxemia must be estimated and emphasis directed at aiding and encouraging the recovery of injured cerebral and cortical tissue If the period of circulatory arrest was less than three minutes complete recovery may be expected but if more than eight minutes survival is not likely

In the presence of sudden circulatory arrest in action and indecision will certainly lead to fatality Just as artificial respiration is mandatory for respira tory failure so must cardiac resuscitation be quickly and effectively instituted for circulatory arrest and if necessary supplemented by the considered and ju dicious administration of procaine procaine amide hydrochloride epinephrine quinidine or electric stim ulation of the heart

ANGINA PECTORIS

Angina pectoris is a descriptive term applied to attacks of paroxysmal pain or discomfort in the chest ordinarily induced by exertion or emotional upsets and promptly relieved by termination of the precipi

ating factor The pain is classically substernal and may or may not radiate beyond this area Radiation is most common to the inner aspect of the left arm occasionally as far as the finger tips Radiation to other areas often occurs and occasionally the pain arises in one of the referred areas The attack seldom lasts longer than five minutes If the attack persists beyond 15 minutes coronary insufficiency or coronary thrombosis instead of angina pectoris should be sus pected Anxiety and fear of impending death often accompany angina pectoris while diffuse sweating and dyspnea are unusual

TREATMENT The immediate attack is treated by nitrites Nitroglycerin tablets 0 3 0 6 mg (1/200 1/100 gr) sublingually are usually effective Amyl nitrite by inhalation usually brings prompt relief Either drug may diminish arterial tension or cause severe headache and palpitation Either drug may be repeated if relief does not occur but overuse is to be avoided In general these drugs are ineffective or even harmful when used in the attempt to relieve the pain of coro nary insufficiency or coronary thrombosis

The frequency of anginal attacks sometimes can be diminished and the exercise tolerance improved by use of longer acting vasodilator agents There are many such preparations available but papaverine and drugs of the xanthine series have enjoyed the longest use It is generally believed now that pentaerythritol tetranitrate (Peritrate®) 10 to 20 mg 4 times daily is more useful The recent introduction of a delayed

action preparation of Peritrate® 80 mg at 12 hour intervals shows promise of becoming a useful drug Ethyl alcohol has no significant effect on the coronary circulation and its ability to relieve the pain of angina pectoris appears to be based entirely on a sedative action

MYOCARDIAL INFARCTION

One of the common emergencies with which the physician is confronted is the patient with an acute myocardial infarction who requires immediate emergency therapy

TREATMENT The measures used in treatment are dictated by the severity of the symptoms and signs associated with myocardial infarction The initial therapy is directed toward supplying oxygen proper handling of the patient relief of pain and combating shock if it exists The less immediate therapy is designed to prevent thromboembolic complications to forestall the development of dangerous cardiac arrhythmias and to assure the prompt recognition and treatment of cardiac failure if it appears

Oxygen can be supplied on an emergency basis by the methods outlined in Chapter 2 It should be remembered that in small communities and farming areas oxygen can be obtained from welders and farmers that is quite suitable for use in emergencies If oxygen is not immediately available prompt and vig

orous attempts should be made to procure it and con sideration of its use should not be abandoned merely because a well organized oxygen therapy service is not at hand

The patient should be gently handled and he should be kept warm The optimal position is the semierect or Fowler s position This position reduces venous pressure at the heart and aids pulmonary ven tilation If the patient is unconscious this position is likely to increase the tendency to obstruction of the air way and the various means for controlling a patent airway outlined in Chapter 1 should be immediately applied Especially when extra oxygen is not avail able airway obstruction should not be allowed to de-velop or persist because it may significantly reduce the oxygen supply to a heart already suffering from oxygen deficiency

These seem like petty details but they are impor tant with respect to the patient s welfare and chances for life

MORPHINE The relief of pain in coronary occlusion is paramount and for this purpose mor phine is the drug of choice It not only relieves the pain but induces rest thereby diminishing the patient s oxygen demand However profound narcosis is to be avoided Morphine may be administered by either the subcutaneous or the intravenous route

Intravenous administration of morphine—If a prompt effect is desired morphine should be given intravenously Pain is relieved almost immediately

but fully as important is the fact that intravenous administration permits evaluation of the effect of the drug as it is being given and its discontinuance when the desired effect is obtained There is less tendency in these circumstances to overdose the patient The drug should be given over 3 to 5 minutes, preferably in about 5 cc of saline or distilled water although it can be administered in the same preparation that is given subcutaneously When the latter preparation is used administration is conveniently delayed by withdrawing blood into the syringe at frequent intervals during the injection Pain is usually relieved by less than 15 mg (¼ gr) if not more can be given by the intravenous route until the desired relief is experienced

Intravenous administration of morphine prevents the harassing situation experienced by many a doctor who in an emergency has administered morphine subcutaneously only to find the patient still writhing in pain 15 or 30 minutes later He is then tempted to give more morphine because of the patient s complaint and the insistence of the family At this point he does not know whether the patient needs 8 mg (⅛ gr) 15 mg (¼ gr) or even 30 mg (½ gr) He usually gives an additional 15 mg This may relieve the pain but it may also depress the respiration The impaired ventilation often accompanied by respiratory obstruction associated with relaxation of the jaw due to narcosis from the morphine seriously hampers oxygenation especially when oxygen therapy has not yet been started

For re emphasis there is no contraindication to

the use of morphine intravenously when it is properly administered

Morphine is not essential if the pain is relatively mild It can easily be relieved by meperidine (Demerol®) or codeine If the pain already has subsided by the time the patient is seen none of these drugs is necessary

BARBITURATES If there has been no pain or if it already has subsided sedatives often are advisable A number of barbiturates can be administered intravenously in this emergency situation Amytal Sodium® phenobarbital sodium (Luminal®) and pentobarbital sodium (Nembutal®) Amytal® and phenobarbital are given in 10 per cent solutions and pentobarbital in a 5 or 6 per cent solution The drugs are given slowly over a 5 minute period and/or until the patient begins to fall asleep and are then discontinued Ordinarily intravenous preparations are unnecessary and phenobarbital 30 mg 3 or 4 times daily suffices Sedatives do not relieve pain and should not be given for this purpose

VASODILATOR DRUGS The use of vasodilator drugs during the initial stages of treatment is less common today than in the past They should not be used if there is evidence of shock However many physicians through long usage have found that certain vasodilators when used with morphine are effective in relieving the pain of acute myocardial infarction The vasodilator most often used for this purpose is aminophylline Ten to 20 ml of a 2 5 per cent solution

is injected very slowly intravenously Few if any, side effects follow intravenous administration of aminophylline if the injection is made slowly With too rapid injection arterial tension may fall precipitously

Shock with Myocardial Infarction

Significant and prolonged shock is a critical sign and upward of 80 per cent of these patients die unless the shock can be corrected Theoretically the slow intra arterial infusion of small amounts of whole blood would seem indicated but significant success has not followed its use Several vasopressor sympathomimetic amines are more useful The preferred drugs are norepinephrine (Levophed®) mephentermine (Wyamine®) methoxamine (Vasoxyl®) and phenylephrine (Neosynephrine®) Most of the experience has been with nor epinephrine (Levophed®)

Nor-epinephrine (Levophed®) can be easily controlled by intravenous drip administration for the variable periods of time that are necessary Care must be taken to avoid extravascular infiltration of the drug to prevent severe local necrosis Ordinarily from 4 to 8 mg are added to a liter of isotonic glucose or physiologic saline solution and the rate of flow is adjusted to maintain the systolic blood pressure at about 100 mm Hg in most patients or at a somewhat higher level in those patients known to have been previously hypertensive

The other sympathomimetic amines also can be

administered intravenously but they are chiefly used to initiate therapy because they can be administered intramuscularly while preparations are being made to administer nor epinephrine (Levophed®) intravenously Mephentermine (Wyamine®) 15 to 30 mg or phenylephrine (Neosynephrine®) 5 to 15 mg are effective by the intramuscular route

It is to be emphasized that the adjunct measures of *rest relief of pain and the administration of oxygen are paramount in the treatment of cardiac shock*

ANTICOAGULANT THERAPY A detailed discussion of anticoagulant therapy in acute myocardial infarction is beyond the scope of this chapter Nevertheless several points are worthy of emphasis There is no unassailable evidence that every patient with myocardial infarction requires anticoagulant therapy There is evidence for its need in those patients who for one reason or another are severely ill from the out set the so-called poor risk patients Anticoagulant therapy requires strict and reliable laboratory control and if it is to be used it should be begun as soon as possible after the myocardial infarction

Heparin is commonly used for the emergency and it may be administered as follows intermittent intra venous injection of 50 75 mg every 4 to 6 hours intramuscular injection of 100 mg of a concentrated aqueous solution every 8 hours intravenously by slow drip adjusted to administer approximately 300 mg in 1 liter of isotonic glucose solution in 24 hours or a long acting preparation containing 200-400 mg

injected deeply intramuscularly once or twice each 24 hours In any case dosage is adjusted to prolong and maintain the coagulation time to between 30 and 60 minutes by the Lee White method

Should hemorrhage occur the heparin effect can be quickly reversed by the transfusion of fresh whole blood or by the intravenous infusion of protamine sulfate 2 mg per kg diluted in 500 ml of isotonic sodium chloride given slowly over at least 2 hours

Polybrene probably 1 5 Dimethyl 1 5 diazaunde camethylene polymethobromide has been more recent ly introduced and is believed to be superior to protamine sulfate as a heparin action neutralizer A recommended intravenous dose is 0 7 mg of poly brene to 1 mg of heparin If insufficient it may be increased to a total dose of 1 1 The polybrene 1 1 5 mg per kg of body weight should be diluted to 100 ml in isotonic sodium chloride or 5 per cent dextrose in distilled water and allowed to flow at a rate varying from 80 to 100 drops per minute The administration is over a 10 15 minute period Neutralizing effects should be evident within 5 minutes

Several anticoagulants are now available for the prolonged effect necessary during the required 3-4 weeks of therapy The experience which the physician has had with a given preparation outweighs the theoretic advantage of one over another Dicumarol® (bishydroxycoumarin) continues to be widely and ef fectively used It is administered orally usually in an initial dose of 300 mg Often 200 mg will be required the second day and 100 mg on the third day Satis

factory anticoagulation is usually attained in 36 to 72 hours after which heparin may be discontinued Maintenance requirements usually fall within the range of 50 100 mg daily All doses of Dicumarol® initial and maintenance are gauged by the daily prothrombin determination Efforts are made to maintain the prothrombin time at from 2 to 2½ times the control value If the prothrombin time becomes excessively prolonged or if hemorrhage occurs Dicumarol® is discontinued and if necessary vitamin K1 (Mephyton®) is given intravenously in a dose of 5 50 mg The smallest effective dose is advisable The latter preparation is available for oral administration but it is less effective

CARDIAC ARRHYTHMIAS

Most of the important cardiac arrhythmias might be considered emergencies but discussion of heart disease in general is beyond the scope of this text Therefore only a few acute disturbances will be mentioned

TACHYCARDIA

When one is confronted with a patient who has very rapid regular tachycardia paroxysmal atrial tachycardia A V nodal tachycardia paroxysmal ventricular tachycardia and atrial flutter must be considered The electrocardiogram is the most exact means of

Mecholyl® norepinephrine (Levophed®), methoxamine (Vasoxyl®) and phenylephrine hydrochloride (Neosynephrine®) also are known to be effective in terminating paroxysmal atrial tachycardia However, their use is limited either by undesirable side reactions or by inherent dangers when given for this purpose to patients with organic cardiac disease

Special mention is made of paroxysmal atrial tachycardia with block which occurs in patients who receive digitalis It is often related to potassium depletion and the administration of potassium salts is the treatment of choice

PAROXYSMAL VENTRICULAR TACHYCARDIA

Patients with paroxysmal supraventricular tachycardia are less likely to have serious organic heart disease than are those with paroxysmal ventricular tachycardia Furthermore the prognosis for the latter is more serious because of its close relation to ventricular fibrillation It is therefore important to recognize this type of tachycardia and to institute treatment promptly

TREATMENT Quinidine sulfate is the drug of choice If the situation is not one of great urgency it may be given orally in an initial dose of 0 4 to 0 8 Gm and under observation 0 4 Gm may be repeated every 2 hours for 5 doses Not infrequently paroxysmal ventricular tachycardia presents an overwhelm

ing emergency and the intravenous administration of quinidine is unhesitatingly resorted to Quinidine gluconate (0 8 Gm in 10 ml ampule) is diluted to 20 ml with isotonic sodium chloride solution and ad ministered at the rate of 1 0 ml (40 mg) per minute It is advisable to observe a continuous direct writing electrocardiographic tracing during the administration and the drug is discontinued upon restoration of nor mal sinus rhythm or before if the QRS complex be comes noticeably widened

If quinidine fails or if the patient is sensitive to the drug procaine amide hydrochloride (Pronestyl®) can be given intravenously at a maximum rate of 200 mg per minute The blood pressure is taken each 30-45 seconds and a significant fall i an indication to diminish the rate of injection A continuous electro cardiographic tracing is advisable here also

Atrial Flutter

The emergency treatment of paroxysmal or estab lished atrial flutter is rapid digitalization for the pur pose of increasing the degree of A V block thereby slowing the ventricular rate The cardiac glycosides serve admirably in this situation the undigitalized patient can be given lanatoside C (Cedilanid®) 1 6 mg intravenously Digitoxin or digoxin (Lanoxin®) may be given orally in the usual dosage if the situation is less urgent

caused by ventricular fibrillation It has been demonstrated that the Adams Stokes attacks sometimes appear when an incomplete A-V heart block changes back and forth to a complete block In these circumstances sufficient digitalis to produce and maintain a complete A-V block may be tried but in general it has been disappointing

PAROXYSMAL CARDIAC DYSPNEA WITH OR WITHOUT PULMONARY EDEMA

Paroxysmal cardiac dyspnea (acute left ventricular failure cardiac asthma) is often a real medical emergency particularly when it is characterized by frank pulmonary edema The patient relieves himself of the ordinary attack which characteristically occurs nocturnally simply by sitting up in bed

TREATMENT Severe attacks often require active therapy The patient s head should be elevated and he should promptly receive oxygen by methods outlined in Chapter 2 With acute overwhelming pulmonary edema it is often beneficial to give oxygen under about 40 mm Hg pressure with an ordinary anesthetic face mask or one of the special masks designed for the purpose This measure of control is reserved for use in the excessive and acute phase of pulmonary edema and should not be used to control the common pulmonary congestion The restlessness and cough respond to morphine given subcutaneously or intravenously (p 85) Blood can often be

promptly although temporarily pooled in the periphery by application of tourniquets high on the four extremities They are applied so that the venous but not the arterial flow is impeded and after about 5 minutes they are alternately released and reapplied in sequence at 5 10 minute intervals When the tourniquets are ineffective phlebotomy is indicated This should be done rapidly, with a large bore needle withdrawing 400 700 ml of blood in 10 15 minutes Atropine sulfate in doses of 1 2 mg (1/50 gr) intravenously has been used Aminophylline 500 mg (8 gr) given slowly by intravenous injection has been reported by many physicians to be effective Finally, if the patient is not already digitalized emergency digitalization may be indicated In this acute situation strophanthin or ouabain injected intravenously in a dose of 0 25 0 5 mg (1/250 1/120 gr) often brings great improvement The dose may be repeated in 12 hours if necessary, but it is preferable to begin digitalization promptly with one of the longer acting preparations so that it will become effective by the time the transient effect or strophanthin or ouabain is wearing off The digitalis glycosides are useful because of their more rapid and often more predictable effect

CEREBROVASCULAR ACCIDENT

One of the more frustrating emergency situations in which the physician often finds himself is the call

to the patient who has recently suffered a stroke The family although they may have been warned of such an occurrence are nevertheless usually agitated over the alteration of their loved one from a vigor ous active alert person to a slobbering gurgling half paralyzed semiconscious or totally unconscious, imbecile appearing individual Unfortunately the phy sician is incapable of doing much to alleviate this distressing situation but he can do certain things to minimize the possible development of atelectasis, pneumonia cystitis etc and to keep the family busy

TREATMENT The physician can well afford to busy himself and the relatives with control of the airway by the measures outlined in Chapter 1 Suc tion of the upper air passages is a positive and bene ficial act as the first order of business The position in which the patient is placed is determined largely by the facility with which the airway can be main tained and the means available for airway control He may be put in the horizontal position either supine or perhaps preferably in the lateral position or it may be advisable to place him in a slight head up position

It is difficult to determine, when first seeing the patient whether the stroke is due to hemorrhage thrombosis or embolism It is well to assume that bleeding has occurred and may be continuing even though it is ultimately proved that there is softening of the brain from infarction In an attempt to stop

the bleeding apply an ice bag to the head and warm the lower extremities by hot water bags or electric pad to divert blood from the head Drugs are of no value for internal bleeding and stimulants are contraindicated

If it can be established fairly definitely that the stroke is due to a thrombus there may be merit in performing a stellate ganglion block on the side opposite the paralysis Although the benefits of this therapeutic technic are debatable there have been many reports of dramatic improvement after the block

Drug therapy —Whatever the cause of the stroke, no immediate drug therapy is indicated unless sedatives are necessary *In no circumstance should the patient be given morphine* If the physician knows that the condition was caused by embolism or thrombosis oxygen may be used to advantage to reduce the extent of permanent damage by improving the oxygenation of tissues in the periphery of the infarcted area

The subsequent paralysis and other sequelae impose many therapeutic problems considered in Chapter 9 on the care of the comatose patient

Again it is well to point out that the analeptic drugs have little value If the patient is in extremis these drugs may be given to impress the relatives, but they are otherwise contraindicated

It is seldom necessary to treat the mild hypotension that may be associated with stroke"

PULMONARY EMBOLISM

When an episode of pulmonary embolism occurs assisted or artificial ventilation with oxygen in high concentration is the most valuable immediate therapy Since there is probably a widespread radiation of autonomic reflexes affecting the heart pulmonary vascular tree and bronchi which are predominantly vagal in origin the use of atropine and also a smooth-muscle antispasmodic such as papaverine is indicated Atropine, 0 8 mg ($\frac{1}{75}$ gr) may be given intravenously and repeated in 10 15 minutes if the first dose has not caused flushing and pupillary dilatation Papaverine is given intravenously in doses of 30 mg ($\frac{1}{2}$ gr) The atropine papaverine administrations may be repeated 3 or 4 times a day

Although it may seem heroic and contraindicated ether by inhalation to the stage of complete anesthesia may appreciably reduce the widespread vasospasm Trendelenburg s operation may be considered

SHOCK

Varying degrees of hypotension often are predominant or at least significant components of many emergency situations It is imperative for the welfare of the patient and restoration to a reasonably efficient circulatory status that the physician recognize the signs of shock and be prepared to treat it promptly and effectively Morbidity and mortality are in direct

proportion to the length of time the patient is in shock

A convenient and practical definition of shock of all types follows Shock is an abnormal physiologic state characterized by circulatory insufficiency caused by a disparity between the circulating blood volume and the vascular capacity

The blood pressure is maintained by the cardiac output and by the peripheral resistance of the arteriolar bed Fall in pressure may be due either to a decrease in peripheral resistance following dilatation of the arterioles and capillaries or to a decrease in cardiac output following a loss of blood volume or following a trapping of the circulating volume in the peripheral vascular bed

Shock may be divided into two types (1) neurogenic shock and (2) traumatic or hemorrhagic shock Treatment varies with type of shock involved

Neurogenic Shock

Neurogenic shock develops as a result of loss of central or peripheral nervous control of peripheral resistance Hypotension occurs when there is a disparity between the circulating volume and the vascular capacity

Cause

The disparity is brought about by a relaxation of the arterioles and capillaries and an increase in the

size of the vascular bed with a concomitant decrease in peripheral resistance The peripheral relaxation may be caused by emotion (fainting) intrathecal or extradural analgesia excitation of certain nerve plexuses (celiac, carotid sinus sacral) drug depression and abnormal positions or abrupt alterations in position Relaxation also may occur as a result of direct action on the vessel walls of toxic products These toxic agents may be from breakdown of tissue or from the action of bacteria One cannot entirely exclude the possibility that some neurogenic hypotension develops from an acute reflexly induced primary cardiac deficiency resulting in diminished output

Characteristics

Characteristics of neurogenic shock are hypotension deterioration in quality of the pulse with little or no change in rate low pulse pressure and pallor although under anesthesia the color change may not be evident There is no hemoconcentration or hemodilution Several features facilitate differentiation of neurogenic shock from the other types of shock In neurogenic shock the history reveals no blood or plasma loss and may indicate a source of reflex initiation of peripheral vascular atonia The pulse pressure is very small and may be so minimal that differentiation between systolic and diastolic pressures is difficult Many times it appears from ausculatory methods that there is no blood pressure Under these circumstances

by reducing the pressure in the cuff of the sphygmomanometer very slowly one may then be able to hear a few pulse beats without being able to differentiate systolic and diastolic pressure. Particularly under inhalation anesthesia the patient may retain a good color and the skin may be warm and dry in the presence of hypotension of neurogenic origin. The pulse rate is usually unchanged from that existing prior to the development of hypotension. The hypotension frequently appears abruptly and disappears abruptly. The most reliable single clinical sign for differentiating neurogenic shock and the other types is the pulse rate. In any patient in whom hypotension develops without associated or prior elevation in the pulse rate, neurogenic shock can be considered seriously. In patients in traumatic or hemorrhagic shock the pulse rate usually increases. Exceptions to this rule are elderly patients in poor condition with minimal cardiac reserve. Neurogenic shock, particularly that arising from toxins, may progress to hemorrhagic shock if the hypotension is of such a degree that hypoxia causes capillary permeability. It is important to treat neurogenic shock in order to avoid tissue hypoxia associated with hypotension.

Treatment

Neurogenic shock is treated specifically by overcoming relaxation of the peripheral vessels. This can be accomplished best by removing the cause. If the

cause is manipulation around a nerve plexus the manipulation may be discontinued, or if it is not possible to avoid the manipulation a local anesthetic can be *injected directly into the plexus to block the reflex* Abnormal positions can be alleviated, and abrupt changes in position avoided If the cause cannot be determined or it cannot be removed one must then employ vasopressor drugs to combat the peripheral relaxation of arterioles and capillaries Vasopressor drugs should be given by vein in small repeated doses until the desired response is secured Ephedrine Neo synephrine® methamphetamine (Methedrine®) and methoxamine (Vasoxyl®) may be used Epinephrine is contraindicated because of the secondary relaxation that accompanies its use The vasopressor drugs should not *be injected subcutaneously or intramuscularly if hypo* tension is severe because absorption will not take place and a response will not be obtained Furthermore if and when a normal tension is restored, the drug injected subcutaneously and intramuscularly during the hypo tension will then be absorbed and an abnormally high pressure will result

It is good policy in neurogenic shock to administer oxygen and lower the patient s head until specific measures have restored normal tension Oxygen helps to reduce the hypoxia and lowering of the head pro motes venous return The head should not be lowered more than 10 15 degrees In mild shock these two simple measures will suffice to restore the blood pres sure

Traumatic or Hemorrhagic Shock

This type of shock includes traumatic surgical and hemorrhagic shock It is a state of circulatory deficiency in which the disparity between circulating volume and vascular capacity is occasioned by a reduction in circulating volume through plasma loss the trapping of intravascular fluid in the peripheral vascular bed loss of water or loss of whole blood

Causes

Hemorrhage accounts for the reduced circulating blood volume in most cases Plasma loss occurs in cases of burn crush injury intestinal lesions affecting the circulation of the intestine (obstruction) bile peritonitis pancreatitis pneumonia bacteremia and pulmonary edema Water loss occurs in cases of severe and rapid dehydration

Characteristics

The characteristics are pallor hypotension with moderate reduction in pulse pressure increase in pulse rate and reduction in pulse quality perspiration cold skin anxiety and restlessness The pallor and cold skin are due to a constriction of the peripheral arterioles in an attempt to reduce the vascular capacity and therefore the circulatory disparity inherent in shock The

anxiety and restlessness are due to associated tissue hypoxia Hemoconcentration develops when there is loss of plasma or dehydration without loss of the blood elements The plasma loss occurs at the site of trauma and usually the volume is sufficient to precipitate shock Patients developing shock after trauma usually have associated whole blood loss Shock which appears in patients exposed to toxins, hypoxia, etc is presumably due to loss of circulating volume from trapping of large portions of the circulating fluid and its formed elements in hyporeactive capillary beds The clinical signs that have been outlined appear relatively late in the development of shock This is particularly true of the blood pressure and one must be aware that a fall in blood pressure means that the patient is approaching the irreversible phase The most reliable early clinical sign of shock is an increase in the pulse rate and constant and close observation of it and the blood pressure is imperative

It is pertinent to point out that one must correlate all the signs of shock in order to evaluate the type and degree and the progress of the shocked state It is important not to overlook the patient s color the degree of perspiration and the temperature of the skin These simple observations are a tremendous help in diagnosis and therapy of shock It is also important whenever possible to know the patient s normal blood pressure and pulse rate Many patients have normally low pressure and are not in shock with systolic tension as low as 80 mm Hg Other patients have normally high blood pressure and may be in shock with systolic

pressure of 120 mm Hg Trends in the curves of pressure and pulse rate will assist materially in the early recognition of shock and the detection of improvement following therapy

One of the most exasperating circumstances is that in which a hypotensive state has developed which from the available history and signs, appears to be surgical shock With appropriate fluid replacement there is a response in the blood pressure which however is limited and below the patient s original level With replacement of fluids in amounts which with conservative estimates seem to be greater than the loss there is no improvement One is then confronted with the problem of determining whether or not there is an element of neurogenic shock an incorrect estimate of the loss or cardiac decompensation One is reluctant, particularly in the elderly patient with a borderline cardiac reserve to continue fluid replacement and is at the same time unwilling to permit the residual hypotension to persist without therapy

In this situation additional information can be secured by the use of methods for determining plasma volume

Treatment

Treatment is directed toward restoring the circulating volume Among the fluids which are effective in performing this task are blood and plasma Saline and glucose solutions are not effective in replacement therapy because except in early shock they pass

through capillary walls and do not remain in the circulation In most surgical and traumatic cases the patients lose sufficient whole blood to require transfusion Patients in shock from loss of plasma through burned surfaces are benefited by the restoration of plasma but these patients develop secondary anemia and require transfusion It is a rare patient who develops shock without blood loss, and from a practical standpoint blood transfusion is preferable to transfusion of plasma Plasma is useful for emergency replacement until blood can be obtained for transfusion

There have been many attempts to develop satisfactory blood substitutes such as gelatin solutions and acacia None has proved uniformly successful However a product known as dextran which contains large molecule polysaccharides has found a useful place as an emergency substitute for plasma or blood It is emphasized however that although this preparation usually succeeds in elevating the blood pressure and in maintaining it for appreciable periods without deleterious effects it does not carry the vital elements present in plasma nor is it capable of transporting oxygen As a consequence it should be used only as an emergency replacement fluid It should be remembered that restoration of a normal blood pressure does not necessarily represent a restoration of all the physiologic deficiencies associated with shock

It should not be overlooked that patients may be in shock from severe dehydration and that restoration on an emergency and acute basis by using saline solution is indicated

Oxygen must be given because hypoxia is a potent factor in the initiation or perpetuation of shock

It is advantageous to place the patient in moderate headdown position The patient should be kept warm, *but* it is poor practice to overheat the patient by wrapping him in hot water bottles and innumerable blankets because the perspiration and dilatation of peripheral capillaries induced by this practice promote further loss of fluid and prolong the period of shock

Anesthesia should be lightened if shock appears in a patient under inhalation anesthesia This enables the patient to compensate somewhat by constriction of his peripheral vascular bed Deep anesthesia promotes relaxation of the peripheral vessels

The use of vasoconstrictive agents such as ephedrine phenylephrine (Neosynephrine®), methoxamine (Vasoxyl®) methamphetamine (Methedrine®) and especially norepinephrine (Levophed®) as a temporary expedient to elevate and maintain a functional arterial tension until adequate replacement therapy can be instituted may be justified It has been and is still argued that the use of these drugs in surgical or hemorrhagic shock is contraindicated because peripheral vasoconstriction is already intense and these drugs may serve only to enhance ischemia and tissue hypoxia It is also argued that the drugs may contribute to the development of kidney insufficiency by constriction of glomerular vessels Nevertheless there is evidence mostly of an uncontrolled clinical nature which seems to indicate that circulation may be improved without serious augmentation of peripheral tissue hypoxia by using these drugs

in the presence of shock It is very important, however, that they be used only on a temporary basis until suitable replacement therapy has been effected It must not be assumed that restoration of a satisfactory blood pressure with vasopressor drugs is adequate therapy

Nor epinephrine as a continuous intravenous drip in a 1 200 000 solution has achieved tremendous popularity The drug is undoubtedly a powerful vasopressor and is capable of restoring arterial tension in greatly deteriorated circulations It is useful as a temporary expedient as mentioned above and it may be helpful in relatively long standing neurogenic shock arising from central nervous system deficiencies However it has serious disadvantages Local ischemia and necrosis at the site of injection is not uncommon In addition it is sometimes difficult to wean a patient after long administration There is little evidence to indicate that this preparation, except in short term (hours) shock has saved lives There is evidence that whereas nor epinephrine constricts glomerular vessels methamphetamine does not Perhaps methamphetamine would be a better drug to use in the circumstances mentioned than nor epinephrine although it appears to be less effective in relieving acute and severe hypotension

It is important to reiterate that elevation in pulse rate and fall in blood pressure are late signs of shock When these signs appear treatment must be started promptly and must be vigorous until the blood pressure and pulse return to approximately normal limits It is well to remember that the estimated loss of blood is often much less than the actual loss It is important

also to remember that restoration of blood particularly when there has been an acute loss must be prompt and adequate This may mean that, to restore competent circulating volume blood will have to be pumped in Do not depend on gravity flow even through a large needle and do force blood into a failing circulation if the pulse and blood pressure fail to show a response Blood can be pumped into a patient in shock until the blood pressure is within approximately 10 per cent of its original level This can be done in the patient with a minimal cardiac reserve and even in the patient with cardiac decompensation

Arterial transfusion has been recommended as a means of combating acute and severe blood loss There is no question that it is an effective method of restoring blood pressure promptly and there may be a shorter period of reduced coronary flow However the technic requires additional and more complicated equipment and arterial puncture or cannulation is not to be considered lightly

In operations in which acute and severe blood loss may be anticipated such as pulmonary resection and cardiac surgery arterial transfusion may be advantageous However experience has shown that equally effective fluid replacement can be accomplished intravenously if the blood is introduced rapidly

If fluid replacement is thoroughly accomplished (by pumping if necessary) and there is no response in pulse rate or blood pressure one must suspect that the hypotension is due to some other factor The hypotension may be on a neurogenic basis or it may be due to

cardiac decompensation a vascular accident a pulmonary embolus or a coronary thrombosis In a few patients particularly the aged and those with minimal cardiac reserve it is well to keep in mind that refractory hypotensive states may be of cardiac origin As a consequence it is sometimes beneficial to give a therapeutic trial to elevation of the head of the patient (semi Fowler position) The traditional head down or horizontal position for patients with this type of hypotension may precipitate or perpetuate the hypotension In such instances intravenous fluid therapy is of no value and may be detrimental

The adrenal corticoids especially hydrocortisone intravenously have enjoyed popularity in the treatment of shock In some communities this preparation has become almost a panacea for shock Except in clearly demonstrated states of adrenal insufficiency or except in the patient receiving substitute adrenal preparations for therapy there rarely is need for these drugs in the treatment of shock

Rarely does a patient exhibit hypotension which is refractory to fluid replacement and to vasopressor drugs Often there is simply a miscalculation in the amount of blood loss and these patients require only additional blood However if hypotension persists, pallor and perspiration are prominent responses are sluggish and color is poor a dramatic improvement may be achieved with administration of hydrocortisone intravenously One must learn to differentiate the causes and the appearance of the different types of hypotension so that therapy may be specific and beneficial

4

Acute Poisoning

IN AN editorial in the *Journal of the American Medical Association* the statement is made that 3 deaths occur every 24 hours in the United States as a result of accidental poisoning In the same period at least 425 individuals will eat or drink something that neither they nor anyone else intended for human consumption and they will turn up in hospital emergency rooms sick but alive

A large proportion of these poison cases occurs in children and most of these accidental poisonings may be traced to carelessness in the home in allowing easy access to medicines household agents and other poisonous materials Children will put almost anything into their mouths The Committee on Toxicology of the American Medical Association has estimated that there are about a quarter of a million brand name chemical products which may be used in the home in farming and industry While most of these are useful many are a potential source of danger when used improperly or left in easy reach of children As an example cleaning fluids are widely used around the

home and almost all are poisonous if swallowed, while others are poisonous if inhaled

Outside the home another large source of potential poisoning is the multitude of insecticides, pesticides and rodenticides *

A single type of agent *i e* containing the same active or poisonous ingredient is usually marketed under numerous trade names This adds to the problem for the physician in identifying the toxic agent The original packages are usually well labeled but often the package is not available since frequently such materials especially unused quantities are stored in paper sacks jugs pop bottles and similar containers

The treatment of acute poisoning usually presents perplexing problems to the doctor With an ever increasing number of toxic substances to which modern man may be exposed a comprehensive treatise on poisoning should be part of every physician s office reference library

Determination of the agent often is difficult and quick decisions are important The diagnosis is facilitated if the physician has in mind certain basic and characteristic toxicologic signs and symptoms of the commonly used and more potent drugs for example the cycloplegic and drying effects of the belladonna series and the slow respiration and miosis caused by the opiates The conditions under which the treatment must be given may be difficult especially in the

*A large number of trade names with their formulations are annually revised and published in *Pesticide Handbook* State College Pa College Science Publishers

highly stimulated patient Specific antidotes must be readily available and proper dosage well in mind Physical signs and symptoms are often those produced by a large number of drugs and the doctor must combine considerable detective ability with this medical knowledge of poisons He must be able to detect characteristic and definitive clues among the host of symptoms and signs that are common to many poisonings Acquaintance with the active ingredients of common household agents proprietary medicines and the general classes of agents used in the insecticides and rodenticides will often facilitate proper identification of the causative agent in poison cases caused by these types of agents Further the physician should be informed of the medicolegal aspects of toxicology so that he will exercise the proper precautions in cases of suspicious poisoning

Poisoning may be homicidal suicidal or accidental The most common symptoms of poisoning are sudden illness in previously well persons such as nausea vomiting diarrhea tenseness abdominal pain cyanosis depression stupor coma hyperexcitability delirium, convulsions ataxia paralysis circulatory collapse chills fever or subnormal temperature Often the treatment must be purely symptomatic but every effort should be made to identify the responsible poison Although every case of poisoning must be considered by itself certain general principles of treatment are useful Because treatment must be instituted early the doctor must use the antidote nearest at hand and many of these antidotes are common household

materials Removal of the poison, delaying its absorption rendering it harmless and controlling the symptoms are the primary objectives

GENERAL PRINCIPLES OF TREATMENT

Certain helpful and oftentimes life saving measures may be instituted before the physician arrives on the scene Directions for such measures should be given by telephone as soon as the emergency call has been received It is obvious that they must be limited to such measures as a layman is capable of performing In the main such instructions are more or less included under general principles of treatment A good command of the general principles of management is highly important Additional instructions should include keeping the patient warm and preferably at rest in bed The order of the emergency measures to be undertaken will depend on the condition of the victim i e the institution of artificial respiration often may be the first and most important step

If the general emergency measures have not been undertaken before the physician arrives the patient s condition will determine the order in which the steps should be taken If the patient is in a state of shock in convulsions or has markedly depressed respiration the symptomatic treatment must take precedence over such steps as removal of poison delaying of absorption and administration of antidotes

Removal of poisons—The removal of the poison

depends on the site On accessible surfaces the area should be thoroughly washed with water If the poison is water insoluble dilute alcohol may be used An appropriate chemical antidote may be added to the washing fluid for example soap sodium bicarbonate borax and lime water for acids and diluted vinegar or lemon juice for alkalis

When poisons are swallowed empty the stomach by gavage or emesis except in instances of strong corrosives or strychnine poisoning In corrosive poisoning perforations may occur and in strychnine poisoning the stimulation is usually sufficient to set off a convulsion In the latter instance the convulsions must be controlled before lavage is attempted Lavage is the most important method for removal of poisons from the stomach Ordinarily gastric lavage is not indicated unless the patient is seen within the first 4 hours after the poison has been ingested Lavage is best accomplished by the use of the largest lumen tube that can be comfortably passed orally For adults a no 30 and for children a no 20 Fr tube is suitable Very small bore nasal tubes are unsatisfactory for rapid washing of the stomach because they clog easily The tube should be dipped in cold water and inserted carefully to avoid entering the trachea In general the technic of passing a tube is as follows

Lay the patient on his right side with head lower than waist Measure the distance on the tube from mouth to stomach and mark the distance on the tube with a piece of adhesive tape Open the patient s mouth use a gag if necessary pass the tube over the

tongue and towards the back of the throat Do not extend head or neck Be certain that tube is in the stomach

The choice of fluids used in gastric lavage depends on the type of poison swallowed In general ordinary tap water is the wash fluid used however as in phenol poisoning the washing should be carried out with an oil The stomach contents are removed either by siphon action or an irrigating syringe The latter is preferable in children Wash repeatedly in order to increase the total amount of poison removed In children do not use over 50 cc of fluid at each injection since larger amounts may promote passage of materials through the pylorus In adults the washings may be 150 200 cc for each washing

Household remedies which will provoke vomiting are 1 teaspoon of mustard stirred in a glass of warm water or 1 tablespoon of salt in a glass of water Other emetic agents are zinc sulfate 2 Gm (30 gr) or cop per sulfate 0 5 Gm (7 5 gr) in a glass of water How ever the most reliable emesis is obtained by hypodermic injection of apomorphine 5 mg (1/12 gr) After the lavage or emesis give a cathartic dose of magnesium sulfate 15 Gm (½ oz) It should be remembered that vomiting can be produced only if the medullary centers are still responsive In severely depressed and uncon scious patients the emetic drugs will be ineffective

Delay of absorption —When poisons are applied to wounds or injected absorption can be delayed by application of a tourniquet centrally to the poi son or by injection of epinephrine at the site to

produce vasoconstriction When use of a tourniquet is not feasible suction excision and cautery may be tried When gases are the noxious agents removal from the exposure and free ventilation of the lungs using artificial respiration and oxygen is indicated (see Chapter 1) If the gas is an irritant the patient should be watched closely for the development of glottic laryngeal or pulmonary edema Glottic and laryngeal edema may be controlled on an emergency basis by the application directly or by spray of a 5 per cent cocaine solution Pulmonary edema is treated with oxygen under positive pressure (p 98)

Administration of antidotes—Antidotes that render the agent insoluble or slow its absorption should be used in connection with lavage and between emesis

Antidotes against acids are magnesium oxide soap chalk and sodium bicarbonate

Antidotes against alkalis are dilute vinegar and lemon juice

Antidotes against alkaloids are oxidizing agents such a potassium permanganate 0 15 Gm (2½ gr) in a tumbler of water and hydrogen peroxide diluted one half with water Precipitation of the alkaloids may be accomplished by tannic acid 1 teaspoon in a glass of water strong tea and tincture of iodine 10-15 drops in a glass of water

Antidotes against heavy metals are of two types Precipitation of heavy metals may be accomplished by raw egg whites More specific antidotal remedies are sodium thiosulfate and sodium formaldehyde sulfoxylate

When the nature of the poison is unknown one may safely administer the universal antidote

Pulveri ed charcoal Burned toast (2 parts)
Magnesia oxide Milk of magnesia (1 part)
Tannic acid Strong tea (1 part)

In the universal antidote the charcoal acts by its absorptive action the tannic acid precipitates alkaloids certain glucosides and many metals and the magnesium oxide neutralizes acids

Antidotes should be administered for the residual poison not removed by gastric lavage When a stomach tube is used some of the antidote and other remedies should be left in stomach before tube is removed

Physiologic Antidotes

There are only a few specific physiological antidotes available against poisons Some of these are

Dimercaprol (*B A L*) This is a specific against arsenicals, mercurials and gold salts

Ethylenediamine tetra acetic acid (E D T A) The calcium compound of this agent is used intravenously in lead poisoning

Atropine It antagonizes the parasympathetic stimulants such as parathion and T E P P, also toadstool poisoning of the Amanita muscaria

Oxygen Used in the treatment of carbon monoxide poisoning to hasten the breakdown of the carbon monoxide hemoglobin complex The use of oxygen-CO_2 mixture instead of pure oxygen is of doubtful advantage, and may even be harmful

N-allylnorphine HCl (Nalline®) or *L 3 hydroxy N allyl morphinan tartrate (Lorphan®)* Specific antagonists against opiates and potent synthetic narcotics

Sodium nitrite and *sodium thiosulfate* Used for cyanide poisoning

Calcium gluconate or lactate Administered intravenously for oxalates and fluorides

Sodium menadiol diphosphate and *Vitamin K* Used for prothrombin inhibitors bishydroxycoumarin (Warfarin)

Antivenoms Used against snake bites insect bites

Symptomatic treatment —This usually is directed toward maintaining respiratory function and preventing circulatory failure Shock is a common accompaniment of severe poisoning and should be treated as outlined earlier (pp 102 ff) Reflex stimulation of the respiration by the inhalation of aromatic spirits of ammonia alternate application of heat and cold or friction with alcohol may be used This type of stimulation cannot be maintained for any period of time and artificial respiration and oxygen therapy may be necessary The latter is very important if an asphyxiant gas is the cause of the poisoning Lowering of the head to increase blood supply to the brain and reflex stimulation by inhalation of dilute ammonia are temporary measures to be used until more definitive therapy is instituted (Chapter 2) Convulsions are controlled by Pentothal® intravenously ether or chloroform When physiologic antidotes are used they

* Not under Harrison Narcotic Law

should be given parenterally otherwise they are likely to be vomited Overdosage of antidotes must be avoid ed because this in itself may cause poisoning

Specific therapy for some of the more common poisonings follows

LOCAL IRRITANTS AND CORROSIVES

Corrosive Acids

TREATMENT The acids must be quickly di luted with large quantities of water and neutralized with weak alkalis such as soap solution milk of magnesia 100 300 cc (3½-10 oz) calcium hydroxide (lime water) 200 250 cc (6 8 oz) aluminum hydrox ide gel 60 cc (2 oz) Sodium bicarbonate or calcium carbonate are adequate neutralizers but the evolution of carbon dioxide from the carbonates may rupture the corroded stomach After neutralization has been carried out milk egg whites or bland oils such as liquid pe trolatum or butter should be administered for their soothing effect If pain is present morphine should be given

Corrosive Alkalis

TREATMENT Treatment is much the same as for acids, except that acids are used to neutralize the alkali Agents used for this purpose are diluted

vinegar 100 200 cc (3½ 7 oz) lemon juice 100 200 cc (3½-7 oz) hydrochloric acid 0 5 per cent 100 200 cc (3½ 7 oz)

NOTE In severe corrosive poisoning the patient should be hos pitalized and treated for possible stricture

Emptying the stomach by lavage is desirable but may be dangerous if the corrosive action is severe Soothing agents such as those used for acids are ad ministered after the initial treatment Eyes injured with either acids or alkalis should be copiously washed with water or saline and covered with a sterile bandage The patient should be seen by an ophthalmologist as soon as possible

CORROSIVES

Phenol (carbolic acid) and cresol (Lysol®) are popular agents for suicide Lysol® and other saponated cresol solutions used in douches and phenol prepara tions for skin diseases have cause serious poisoning When taken orally the local symptoms are burning of the mouth and throat nausea vomiting and ab dominal pain In addition there are circulatory fail ure and central depression

TREATMENT The local effects should be promptly removed by washing with diluted alcohol (or whisky) and application of an oil dressing

When a corrosive is taken by mouth the stomach should be washed with olive oil until odor of the

poison disappears after which 50 100 cc (2 3 oz) of the oil should be left in the stomach Treatment of the systemic effects is purely symptomatic Keep the patient warm and give saline intravenously to promote diuresis Artificial respiration or oxygen therapy may be necessary Morphine should be given for pain

METALLIC POISONING

MERCURY

Acute mercury poisoning usually occurs from the accidental or intentional ingestion of bichloride of mercury The immediate effects are due to the co agulant irritant and corrosive effect on the mucous membranes There are burning and discoloration of the mouth and pharynx and epigastric pain nausea and vomiting The vomitus often contains mucous shreds If treatment is not instituted immediately the symptoms become progressively worse The large in testine and kidneys become involved within a few days the urine becomes scanty and complete anuria may develop and death may ensue in 5 10 days

TREATMENT Administer milk or raw eggs and evacuate the stomach by lavage Follow this by washing with 5 per cent sodium formaldehyde sulf oxylate solution which is preferable to pure water This agent reduces mercuric salts to the insoluble mercurous form On termination of the washing 250

cc of the 5 per cent solution is left in the stomach

BAL (British anti Lewisite 2,3 dimercaptopropanol dimercaprol) —This is an effective antidote against arsenicals mercury antimony, gold and bismuth but not against lead and selenium It is marketed in 10 per cent solution in oil with 20 per cent benzyl benzoate and is administered intramuscularly In mercury poisoning intramuscular injections of BAL should be started as soon as possible For the average adult 5 mg per kg or about 3 cc of a 10 per cent solution is given followed by half this dose in 2 hours and again in 3 or 4 hours Two such injections are given the second day and 1 the third day BAL has given excellent results when used soon after the poisoning but it cannot be expected to be very effective after a few hours delay

If the patient is seen too late i e after severe kidney damage has developed the usual treatment for shock dehydration and salt depletion is carried out

BAL causes some *toxic reactions* Its intramuscular injection in doses of 3 5 mg per kg causes mild symptoms such as perspiration pain in various regions salivation sometimes vomiting restlessness apprehension weakness quickened heart rate and some rise in blood pressure The symptoms usually subside in 1 to 4 hours Larger doses cause muscle tremors rapid thready pulse convulsions coma and death If convulsions occur they may be combated by barbiturates

When BAL is used to combat metallic poisoning *careful dosage on a weight basis* should be observed

phenomena of carbon monoxide poisoning are those of ordinary anoxia except that cyanosis is not present The skin is pale and the lips are usually bright red Prognosis depends on the duration of exposure with prolonged exposure there may result permanent damage to the brain

TREATMENT Treatment of this condition is usually successful if the heart is still beating Give artificial respiration if needed and administer oxygen or preferably oxygen containing 4 7 per cent carbon dioxide The carbon dioxide oxygen mixture acts in a twofold manner it aids in stimulating the respiration and hastens the separation of carbon monoxide from its combination with hemoglobin In very severe cases bleeding followed by transfusions may be necessary Intramuscular injection of 0 5 1 cc of 1 1 000 epinephrine will help to contract the spleen and throw unpoisoned red cells into the circulation The greatest portion of the carbon monoxide hemoglobin will be decomposed during the first hour under adequate respiration of air or oxygen or oxygen carbon dioxide mixture and it is needless to continue the administration beyond an hour if adequate ventilation has been carried out The only damage from carbon monoxide is the anoxia and thus measures other than those used to overcome the anoxia are useless If coma persists one must suspect brain damage Treatment of the sequelae must be purely expectant

CARBON TETRACHLORIDE

The most frequent cause of poisoning by carbon tetrachloride is its ingestion by children in form of cleaning fluids fire extinguisher fluid or solvent for paint, fats and oils However poisoning may also occur from inhalation of the fumes

TREATMENT When carbon tetrachloride has been inhaled remove patient to fresh air give artificial respiration if needed If the poison is ingested wash stomach copiously with water and administer saline cathartic Avoid all fatty liquids After ridding stomach of poison put patient to bed and keep him quiet If patient is a known alcoholic or poorly nourished he should be hospitalized and more specific therapy instituted against liver and kidney damage

HYDROCYANIC ACID AND CYANIDES

Poisoning by hydrocyanic acid is extremely rapid and death may occur in a very short time Poisoning by ingestion of cyanides also is rapid and usually fatal if the agent is taken in sufficient amount The cyanides cause death from anoxia as they hinder the oxidative processes of the tissues When large doses are taken the individual may become unconscious in a few seconds and die within 5 10 minutes In other circumstances the symptoms are vertigo headache dyspnea and palpitation followed by unconsciousness and convulsions

Because the poison acts so rapidly the diagnosis and treatment must be immediate When large doses have been taken the patient usually dies despite any treatment The diagnosis can usually be made from the characteristic almond like odor of hydrocyanic acid

TREATMENT When *inhaled* treatment is the same as for ingested cyanides except for stomach evacuation

When *ingested* give orally such antidotes as sodium thiosulfate 5 per cent hydrogen peroxide 1 5 potassium permanganate 1 1 000 or animal charcoal and follow by lavage Maintain the respiration artificially by administering oxygen The rest of the treatment is specific and must be carried out very promptly Inject slowly (2 to 3 minutes) intravenously 0 2 0 5 Gm (3 7½ gr) of sodium nitrite dissolved in 10 15 cc of water and follow with intravenous injection of 25 Gm (⅚ oz) of sodium thiosulfate in 50 cc of water The injection should extend over 10 minutes and may be followed in 30 minutes if necessary with half the first doses To institute the therapy more promptly during the time of preparation of the sodium nitrite and sodium thiosulfate solutions start the nitrite therapy immediately by application of amyl nitrite pearls to the nose and mouth Discontinue the inhalation of nitrite at the time the intravenous medication is initiated To combat the fall in blood pressure from the nitrites administration of 10 15 mg (⅓ ampule) of ephedrine may be necessary The purpose of the nitrite therapy is to produce methemoglobin The thio

sulfate converts the slowly released cyanide to thiocyanate If the formation of methemoglobin is too great oxygen therapy may be necessary to combat anoxemia and if extreme blood transfusion may be indicated

A less efficient agent for the formation of methemoglobin is methylene blue Fifty cubic centimeters of 1 per cent isotonic solution is administered intravenously if this agent is to be used

Benzol

Poisoning from benzol results from industrial exposure It may be absorbed from the alimentary tract or skin or as vapor from the lungs The poisoning is usually chronic but may be acute when ingested or inhaled in large quantities The symptoms are restlessness dizziness and muscular in-co-ordination followed by unconsciousness and death from respiratory failure

TREATMENT The treatment is purely symptomatic Lavage the stomach with water and support the respiration

Gasoline and Kerosene

The ingestion of kerosene or gasoline by children is fairly common The mere ingestion of these products is not too serious in itself however their ingestion and aspiration may prove serious Ingestion without

aspiration may cause restlessness, in-co-ordination, vomiting and cyanosis With aspiration the lungs are involved, and rapid and weak respiration weak pulse cyanosis coma and convulsions may develop

TREATMENT Since the amount of these agents is seldom known gastric lavage should be done as a precautionary measure against regurgitation and aspiration When gastric lavage is used, it should be performed early before any narcosis occurs Care should be exercised so that the lavage itself does not precipitate gagging and aspiration Avoid any oily laxatives Emesis is contraindicated because of the danger of aspiration Oxygen therapy may be useful if needed to prevent anoxemia Convulsions may be controlled by barbiturates It is probably well to institute antibiotic therapy to lessen the chances of pneumonia from aspiration The antibiotics will not benefit kerosene pneumonia as such but may prevent bacterial invasion

SULFIDES

Poisoning by sulfides occurs chiefly in chemical laboratories and industry The symptoms vary with the concentration of the gas and death may be extremely rapid after exposure to heavy concentrations Locally the gas is a severe irritant causing conjunctivitis and edema of the eyes and swelling of the mucous membranes of the respiratory tract The serious toxic effects are systemic and include headache

nausea dyspnea and malaise High concentrations produce rapid respiration and convulsions

TREATMENT Remove the patient from exposure and give artificial respiration Oxygen therapy should be used, if available If respiration can be maintained the sulfide will oxidize to the sulfate Because pneumonia may develop the patient should be given penicillin

CARBON DISULFIDE

Carbon disulfide is widely used as an industrial solvent and poisoning by inhalation is not uncommon When high concentrations have been inhaled there is initial excitement followed by unconsciousness and convulsions

TREATMENT This is purely symptomatic Support the respiration and control convulsions by barbiturates

CAMPHOR

As little as 0 75 Gm of camphor may be fatal to a child adults are apparently less susceptible It may be found in the home in various forms

Anxiety excitement delirium and epileptiform convulsions result from excitation of the central nervous system Depression usually follows the excitation phase Death may occur early or as late as 24 hours after the

ingestion the cause of death is either respiratory failure during a convulsive seizure or exhaustion and circulatory collapse

TREATMENT The treatment is largely symptomatic as with other C N S stimulants Gastric lavage or emetics are indicated if the patient is seen before onset of severe symptoms The control of the convulsions should be by a short acting barbiturate (pentobarbital sodium 0 3 Gm [5 gr]) intravenously or ether inhalation Avoid opiates Artificial respiration may be necessary in cases of respiratory failure

Methyl Chloride and Methyl Bromide

These agents are used as refrigerants and may cause accidental poisoning if they escape into the room They are chloroform like depressants of the central nervous system Usually removal from the source of fumes to an uncontaminated atmosphere is sufficient However the patient should be observed for the development of pulmonary edema and treatment for this instituted immediately Moderate convulsions may appear and are controlled as described later (pp 229 ff)

CENTRAL NERVOUS SYSTEM STIMULANTS (CONVULSANTS)

Convulsant poisons may act on any part of the nervous system In less than convulsive doses they

cause symptoms of increased excitability of the respective centers

ANTIHISTAMINICS

The antihistaminics are relatively safe drugs but may be potentially dangerous Many of these agents are sold both over the counter and by prescription for the treatment of allergies and colds The antihistaminic drugs in toxic doses produce a complex of central nervous system excitatory and depressant properties The symptoms that may occur are drowsiness flushing dryness of mouth nausea nervousness dilated pupils headache and confusion Large overdoses may cause cerebral stimulation A few cases of quite severe toxic reactions have been reported with hypotension collapse and unconsciousness

TREATMENT Empty stomach by gastric lavage or emesis follow by a saline cathartic If patient is in coma and respiratory depression use artificial respiration Maintain normal blood pressure (See neurogenic shock p 103) Convulsions are controlled by ether or by a short acting barbiturate (pentobarbital sodium 0 3 Gm [5 gr]) given intravenously

BELLADONNA ALKALOIDS

Atropine and scopolamine poisoning may occur after their therapeutic administration as well as from accidental swallowing Children are especially suscep-

tible to them The eating of jimson weed or berries of belladonna sometimes produces symptoms in children Early symptoms are dryness of the mouth difficulty in swallowing increased body temperature dry hot skin weak and rapid pulse increased blood pressure dilated pupils restlessness confusion in-co-ordination and speech disturbances and delirium In severe cases respiratory and circulatory depression follow the excitatory phase and death is due to respiratory failure

TREATMENT If the drug has been taken orally empty the stomach by lavage If the patient is highly excited the lavage may be done under narcosis After the lavage a saline cathartic is useful in the early stages of the poisoning Later and after the relaxant effect of the drug on the intestine is produced cathartics become less effective

Whatever the site of entry give 5 mg (1/12 gr) of pilocarpine subcutaneously at intervals until the mouth is moist Pilocarpine tends to alleviate some of the peripheral symptoms but has no effect on the central action of these drugs For severe excitement states, administer either Nembutal® or Seconal® 100-300 mg (1½, 3 gr) orally Chloral hydrate 1 2 Gm (15 30 gr) in milk or water or paraldehyde 2 6 cc may be given *Do not* use large doses because the depressant phases of the poison and drug may coincide If the patient is seen in the depressive stage central stimulants such as caffeine 0 6 Gm (10 gr) amphetamine (Benzedrine®) 10 20 mg or desoxyephedrine 5 10 mg may be used Ice bags and

alcohol sponges aid in reducing the fever and making the patient more comfortable

Cocaine and Synthetic Local Anesthetics

Toxic reactions to this series of agents are quite common and are given special consideration in Chapter 5

Nicotine

Nicotine is a potent and rapid poison Poisoning may result from the accidental or suicidal swallowing of the insecticide Black Leaf 40 which is a 40 per cent solution of crude nicotine sulfate Nicotine causes stimulation of both the central and the autonomic nervous systems Nausea salivation abdominal pain vomiting diarrhea headache mental confusion and severe weakness are early symptoms The respiration is stimulated and the blood pressure rises Pupils are at first constricted and later dilated Prostration fall in blood pressure difficult respiration and terminal convulsions ensue and death results from respiratory paralysis

TREATMENT Artificial respiration and emptying of the stomach are of prime importance If respiration can be maintained the poison will be detoxified by the body Therefore respiration is the important function and should be maintained by artificial respiration and oxygen therapy Stimulant drugs are

of no value If the blood pressure is very low, inject 10 15 mg (1/3 ampule) of ephedrine intravenously

In mild poisoning the excitatory symptoms may be controlled by short acting barbiturates—pentobarbital sodium 0 1 Gm (1½ gr) or Sodium Amytal® 0 1 0 2 Gm (1½ 3 gr) by mouth

STRYCHNINE

Poisoning from strychnine in older individuals is usually suicidal Instances of poisoning occur in children who eat cathartic pills containing strychnine Restlessness increased respiration jerking movements and stiffness of neck and face muscles are followed by typical strychnine convulsions

TREATMENT After the convulsions have been controlled by Pentothal® ether or chloroform lavage may be instituted if it is believed that some of the poison still remains in the stomach Potassium permanganate 1 1 000 tincture of iodine diluted 1 250 or strong tea should be used for washing the stomach Keep the patient under observation for several hours Any increased reflex excitability and muscle twitches are indications for further administration of barbiturates

CHLORINATED HYDROCARBON INSECTICIDES

This class of compounds includes Aldrin® Benzene hexachloride (gamma isomer lindane), Dichlorodi

phenyltrichloroethane (DDT) dieldrin difluorodi phenyltrichloroethane (D FDT) dichlorodiphenyleth anol (Dimite) dichlorodiphenyl methyl carbonal (D M C) chlordane heptachlor methoxychlor Neotran® Ovotran® and Toxaphene®

These agents are constituents of many insecticides and may cause symptoms due to ingestion inhalations or absorption through the skin when used as sprays or dusting agents The various solvents especially kerosene benzene or other petroleum derivatives are in themselves toxic agents

These agents vary in toxicity but all are capable of being central nervous system stimulants causing restlessness hyperirritability in-co-ordination muscle spasms tremors and convulsions followed by depres sion Death may occur from respiratory failure DDT appears to be the most toxic at least in animals

TREATMENT The treatment for all the group is similar In case of skin contamination wash all exposed areas thoroughly with soap and water If the material is ingested remove from gastrointestinal tract by gastric lavage and saline catharsis Avoid fats or oils since they tend to increase absorption of these agents

If tremors develop they may be controlled by a long acting barbiturate such as phenobarbital how ever for the treatment of convulsive states a short acting barbiturate is indicated i e pentobarbital so dium In serious poisoning the liver should be protected by large doses of carbohydrates and vitamins of B-Complex A high protein diet is also desirable

Xanthine Derivatives

The toxic effects of *caffeine* occur mainly in the central nervous and circulatory systems and include nervousness insomnia restlessness and excitement sometimes progressing to mild delirium The circulatory effects are tachycardia and extrasystole

Theophylline and *theobromine* do not have the central effects of caffeine Both cause gastrointestinal upsets with nausea and vomiting Deaths have b en reported following intravenous injection of *aminophylline* and when given intravenously it should be administered very slowly because death is sudden Caution in its administration is the only safeguard

TREATMENT The stimulation of caffeine can be adequately controlled by depressant drugs (see Chapter 8)

Rotenone

Rotenone is a constituent of certain insecticides It is soluble in lipids but not in water It has not been known to cause poisoning in man In some animals as little as 60 mg will cause convulsions respiratory depression coma and respiratory failure Dogs will tolerate up to 2 gm per kilogram

DEPRESSANT POISONS OF THE CENTRAL NERVOUS SYSTEM

The characteristic effect of all of these poisons is depression of the central nervous system

Severe *barbiturate* poisoning is very common For treatment see care of acute drug depression (p 238)

For morphine poisoning see Chapter 9 especially page 238

Alcohol

The symptoms of acute alcoholism are well known However the physician should always be sure that the condition is due to alcohol and not to such con ditions as unconsciousness from depressant drugs cardiovascular accidents or fractured skull

TREATMENT In not too severe intoxication by alcohol time and sleep are all that are necessary

Methyl Alcohol (Wood Alcohol)

In general the symptoms of methyl alcohol poison ing are similar to those of ethyl alcohol but the sec ondary effects are much more severe This is believed to be due to the incomplete oxidation of the methyl alcohol There are often cerebral edema optic neuritis and atrophy and also neuritis of other nerves Blind ness may be permanent

TREATMENT Lavage the stomach with 4 per cent sodium bicarbonate solution Give 4 Gm (1 drachm) of sodium bicarbonate every 15 minutes for 4 doses it may be necessary to give it by stomach tube Intravenous alkali therapy should also be given The intravenous administration of 1 L of $\frac{1}{6}$ molar

sodium lactate (Hartman s solution) or 250 cc of freshly prepared 5 per cent sodium bicarbonate may be necessary* Some check on the acidosis should be made and if facilities for determining CO_2-combining power are not available the reaction of the urine may be used as a guide to alkali administration Al kali treatment should be discontinued when the urine is neutral or slightly alkaline Cerebral edema may be treated by intravenous injection of 50 100 cc of 50 per cent glucose removal of cerebrospinal fluid by lumbar puncture and oral administration of 1 2 oz of magnesium sulfate

When the patient is comatose, oxygen therapy should be used to support the respiration Further therapy must be purely symptomatic

ANTIPYRETICS

All of the antipyretic analgesics are depressants of the central nervous system Large doses lead to mental confusion muscular in-co-ordination and coma Overdose is treated as outlined in Chapter 9

ACETANILID ACETOPHENETIDIN

Acute poisoning by use of these agents is rare The outstanding symptom is cyanosis which is due to the

*Sodium bicarbonate for intravenous administration requires special preparation because it cannot be sterilized by boiling The water is boiled and the sodium bicarbonate added after partial cooling.

formation of methemoglobin and sulfhemoglobin

TREATMENT Wash out the stomach and administer ½ oz of magnesium sulfate If cyanosis is severe blood transfusions may be necessary Artificial respiration and inhalation of oxygen are helpful Administer sodium bicarbonate orally If shock develops treat as outlined on page 107

Antipyrine Aminopyrine

Cyanosis seldom occurs with poisoning from these agents because the red cells are not often affected Acute poisoning is very rare If convulsions occur they may be controlled by the barbiturates

Salicylates

These agents rarely cause symptoms that require any therapy other than withdrawal or reduction of dosage Suicidal attempts have been reported with acetylsalicylic acid using 20 30 Gm (300–450 gr)

Reactions to acetylsalicylic acid are quite common in allergic individuals and patients should be questioned regarding sensitivity before the drug is prescribed For sensitive patients who require salicylate therapy use sodium salicylate

If angioneurotic edema and other allergic phenomena develop after administration of acetylsalicylic acid treat as for allergic reactions (Chapter 7)

PARASYMPATHETIC EXCITANTS

The organic phosphate insecticides have caused poisoning and death among those working in chemical factories and among agriculturists using them as dusts and sprays Such agents as arecoline choline derivatives neostigmine physostigmine pilocarpine di isopropyl fluorphosphate and octamethyl pyrophosphoramide are used medicinally and overdosage may occur Physostigmine neostigmine and the organic phosphates produce their effects primarily by inactivating the cholinesterase In severe poisoning with these agents death may occur within 2 hours

Diagnosis of this group is based on the triad of colic miosis and profuse perspiration

TREATMENT Give atropine intravenously 0 5 Gm /$^{1}/_{120}$ gr or 1 mg subcutaneously ($^{1}/_{60}$ gr) every 15 minutes until pupils dilate the heart rate is increased and the colic relieved

Phosphate Esters Organic Insecticides Chlorthion Dimeton Diazinon, Malathion Parathion and TEPP These agents are widely used by farmers florists and commercial sprayers

Symptoms are mainly due to their cholinesterase inhibiting action which allows an accumulation of acetylcholine Gastrointestinal disturbances sweating salivation miosis (this may not be seen early) involuntary defecation and urination muscle weakness and generalized convulsions are the most frequent symptoms

TREATMENT IN SEVERE CASES Give artifi cial respiration Administer 2 mg ($\frac{1}{30}$ gr) atropine sulfate intravenously as soon as cyanosis is overcome Repeat every 10 15 minutes until signs of atropiniza tion appear

If poisoning is due to contact the patient must be decontaminated quickly Wearing rubber gloves re move the patient s clothing and bathe patient thor oughly with soap and water A mild alkaline wash is preferable If the poison has been ingested stom ach may be emptied by emesis if the patient is re sponsive otherwise use gastric lavage Treat symp tomatically Atropine does not protect against muscular weakness Watch the patient constantly since the need of artificial respiration may appear suddenly

In less severe cases keep the patient fully atro pinized by 1 2 mg ($\frac{1}{60}$ $\frac{1}{30}$ gr) every hour The drug may be given intramuscularly but the intravenous route is more rapid As much as 10 20 mg (~3 gr to 1$\frac{1}{3}$ gr) per day may be necessary Decontamina tion or emptying stomach are carried out as above Respiration should be watched

Avoid all respiratory depressants such as morphine barbiturates or other sedatives

VASOPRESSOR AGENTS

AMPHETAMINE (BENZEDRINE®)

Moderate doses (5 10 mg) of this drug have been taken over long periods without any apparent ill ef

fects however overdosage leads to irritability insomnia headache, nausea dryness of the mouth anorexia and sometimes diarrhea Cardiovascular reactions including palpitation extrasystole hypertension anginal pains arrhythmias and circulatory collapse may occur The drug may be habit forming Contraindications to and cautions in the use of amphetamine are about the same as those for epinephrine and ephedrine

TREATMENT Oxygen therapy may have value in case of circulatory collapse

DEXEDRINE© D DESOXYEPHEDRINE

These drugs are similar to amphetamine except that they are more potent

EPINEPHRINE

Minor effects of epinephrine are discussed on page 210 Although alarming they have no serious consequence Major accidents with epinephrine usually occur from intravenous injection Cardiac dilatation cerebral accidents, pulmonary edema and ventricular fibrillations are the principal effects and are so serious that little in the way of treatment can be prescribed Contraindications to the use of epinephrine are cardiovascular disease and chloroform and cyclopropane anesthesia The drug should be administered cautiously to patients who have hyperthyroidism Unless death occurs almost at once the toxic symptoms of epinephrine overdose subside quite promptly

Ephedrine

Symptoms of toxic reactions to ephedrine may follow too frequent application in the nose ingestion or the injection of overdoses No fatalities or serious effects have been reported The symptoms of overdosage are nervousness insomnia tremulousness dizziness headache precordial pain palpitation sweating and dilation of the pupils With very large doses there may be a weak pulse and dyspnea in addition

TREATMENT With severe stimulation, sedate the patient with barbiturates orally sodium pentobarbital 100 180 mg (1½ 3 gr) or 8 10 mg of morphine

Digitalis

Accidental ingestion or overdigitalization may produce a number of symptoms including loss of appetite nausea vomiting diarrhea visual disturbances and headache The pulse is usually very slow but there may be tachycardia In fatal poisoning ventricular fibrillation is the cause of death Occasionally the only symptom in children is coma

TREATMENT If the drug is accidentally swallowed or an overdose is taken by mouth lavage the stomach with water Give plenty of fluids by mouth Enforced bed rest until the clinical symptoms disappear is usually all that is necessary

In instances of ventricular tachycardia administer 10 cc of 10 per cent magnesium sulfate intravenously

followed by quinidine sulfate 60-120 mg (1 2 gr) orally every 3 hours for 2 or 3 days

Patients taking digitalis have increased sensitivity *to epinephrine and calcium so these agents are con* traindicated during a digitalis regime

QUINIDINE

This drug can cause symptoms directly and also symptoms that arise indirectly owing to the nature of the disorder for which the drug is used Some indi viduals exhibit an idiosyncrasy to quinidine The re actions likely to occur are respiratory distress cyanosis nausea and vomiting In a patient who is to receive this drug test for sensitivity by administering 0 2 0 3 Gm (3-4½ gr) of quinidine sulfate orally 1 day be fore intensive treatment is started Observe for symp toms

The other possible serious consequences of quini dine are embolism when the heart suddenly stops fibrillating dangerous tachycardia ventricular stand still and ventricular fibrillation Previous digitalization of the patient may aid in preventing some of these serious consequences

ERGOT PREPARATIONS

Acute poisoning is rare and probably occurs only when large amounts of ergot preparations are ingested

to induce abortion The symptoms of acute ergotism are vomiting diarrhea great thirst vertigo dyspnea paresthesias and anesthesias visual disturbances convulsions rapid weak pulse confusion and unconsciousness Treatment is purely symptomatic

Chronic poisoning is more common and probably arises from the injudicious use of ergot preparations Gangrene beginning in the extremities is the most common symptom In addition to the vascular effects headache nausea vomiting and diarrhea confusion depression or convulsions may occur

TREATMENT Withdraw the drug and direct therapy toward maintaining the peripheral circulation Use long-acting nitrites papaverine or aminophylline Sodium nitrite 30-60 mg (1/2-1 gr) may be given orally in repeated doses Erythrityl tetranitrate 15-30 mg (1/4-1/2 gr) papaverine 30-80 mg (1/2-1 1/3 gr) and aminophylline 0 2 Gm (3 gr) may be given orally Nausea and vomiting may be controlled by atropine 0 5-1 mg (1/120-1/60 gr) subcutaneously

ERGOTAMINE DIHYDROERGOTAMINE

These agents are rather widely used in treatment of migraine and may produce all the symptoms of ergotism For severe symptoms the treatment is the same as for ergot poisoning

Posterior Pituitary Extracts

After excessive injection of posterior pituitary ex tracts there may occur striking facial pallor increased peristaltic activity nausea abdominal cramps and in women menstruation like cramps There is no specific treatment for overdose of posterior pituitary extract The patient should be treated symptomatically with oxygen vasodepressor drugs and sedation if necessary

Nitrites

Poisoning by the nitrites may occur from accidental swallowing or industrial exposure There are marked flushing of the skin fall in blood pressure, cyanosis methemoglobinemia and dyspnea Fainting and respir atory failure may occur

TREATMENT Place the patient in recumbent position with the head lowered To combat excessive low blood pressure inject 10 20 mg (1 2 ampules) of ephedrine intravenously Oxygen inhalation is ben eficial If the methemoglobin formation is severe blood transfusion may be life saving

MISCELLANEOUS POISONS

Borates and Boric Acid

Poisoning by borates or boric acid is usually ac cidental The symptoms appear several hours after

ingestion with vomiting diarrhea progressive prostration convulsions and finally shock syndrome

TREATMENT Evacuate the stomach by lavage or emetics and administer Ringer's solution or plasma For shock treatment see page 103

Iodine

The ingestion of iodine causes gastroenteritis abdominal pain vomiting and diarrhea In fatal cases death occurs in about two days from circulatory failure Frequently diagnosis can be based on the iodine staining of the oral tissues

TREATMENT Lavage the stomach with a thin starch paste and continue the washing until all traces of iodine are removed Five per cent sodium thiosulfate may be used instead of the starch solution Because large amounts of fluid are lost in the vomitus and diarrhea saline should be given intravenously If shock arises treat as indicated on page 103

Fluorides

Fluorides are often a constituent of insecticides and acute poisoning may occur from accidental ingestion The early symptoms are those due to the local action in the gastrointestinal tract—nausea vomiting abdominal pain and diarrhea The systemic effects are

central involving the heart Convulsions are common Death is due to respiratory and cardiac failure

TREATMENT Empty the stomach by lavage or emetics Administer soluble calcium salts such as lactate chloride or gluconate Calcium chloride or gluconate intravenously may be of some value General treatment is symptomatic and supportive

OXALATES

Poisoning by oxalates is usually accidental If oxalic acid was ingested the first symptoms are those of a caustic acid Rapid collapse with very weak pulse sometimes preceded by convulsions is the usual course

TREATMENT The specific treatment for oxalates is calcium Lavage the stomach with solutions of calcium salts any soluble salt being suitable Follow with a saline cathartic and large quantities of water Calcium gluconate or lactate should be given intravenously for the systemic effects It is important to stimulate diuresis by administration of fluids in order to prevent deposition of oxalate crystals in the kidneys The patient should be kept on a high carbohydrate protein low fat diet and an increased fluid intake

PHOSPHORUS

Poisoning by phosphorus occurs from accidental swallowing or the inhalation of fumes in industry

The early symptoms are related to the gastrointestinal tract—burning nausea vomiting and diarrhea The vomitus has a strong gastric like odor and is luminous Shock may occur with the patient dying rapidly If death does not occur the patient has a symptom free period of 1 or 2 days The after symptoms are due to effects on the liver heart glands and muscles and there are protracted emesis jaundice oliguria and cardiovascular collapse

TREATMENT When phosphorus is ingested lavage the stomach and administer copper sulfate 0 25 Gm in a glass of water Potassium permanganate 1 1 000 or hydrogen peroxide may also be used After administration of the antidote give magnesium sulfate for catharsis

Local phosphorus burns are thoroughly washed with water or saline and kept moist As quickly as possible after the initial washing apply 1 5 2 0 per cent sodium bicarbonate solution to neutralize any phosphoric acid formed and then wash with a 1 per cent solution of copper sulfate All particles of phosphorus should be removed Subsequent treatment is the same as for any other burn (p 259)

RODENTICIDES RAT POISONS

Some of the newer rodenticides present serious hazards and should be used with utmost caution With increasing emphasis on rodent eradication poisoning will probably be more common

RED SQUILL

This has been used widely as a rat poison It is relatively nontoxic for all mammals that are capable of vomiting

ANTU®, ALPHANAPHTHYLTHIOUREA

This is a reasonably safe agent because it usually induces emesis before toxic amounts are absorbed Toxic absorption may inhibit the normal metabolic functions of the body The sulfhydryl groups seems to be involved

TREATMENT There is no specific therapy Ga vage and oxygen therapy are useful Restrict fluids The use of BAL is contraindicated Cysteine and thio sorbitol seem to have some value

1080® (FLUORACETATE)

This is a very potent rat poison and equally poi sonous to man The substance is very stable has no odor or taste and is easily mixed in bait The toxic effects are chiefly on the heart causing ventricular fibrillation It may also produce central nervous sys tem stimulation

TREATMENT Remove from the stomach by lavage with lime water or another soluble calcium salt Inject calcium gluconate (0 5 Gm as 5 per cent

solution) intravenously and continue a saline infusion The intracardiac injection of procaine hydrochloride has been suggested as a means of preventing the ventricular fibrillation Inject 5 cc of 1 per cent procaine hydrochloride at 2 minute intervals for total of 20 cc Quinidine may also be used Short acting barbiturates such as pentobarbital may be used to combat the central nervous system stimulation

TOXICITY OF ANTIBIOTICS

Penicillin

This antibiotic causes a number of reactions only a few of which are serious Intramuscular or subcutaneous injection may cause pain but usually this can be prevented by the addition of a local anesthetic Mild nitritoid reactions are sometimes seen during intravenous injection and thrombophlebitis may develop if a vein is used too often Allergic reactions occur in about 10 per cent of cases giving rise to rashes angioneurotic edema sometimes muscular twitchings and even convulsions Delayed reactions such as urticaria and pruritus may begin several days after a penicillin regime is started The Herxheimer reaction may occur in syphilitic patients when inadequately pretreated Malaise with chills fever and headache is quite rare

TREATMENT As allergic responses seem to be the most annoying effects the drug should be

POISONOUS SUBSTANCES IN 48 HOUSEHOLD ITEMS*

Polishes and Waxes For Furniture and Floors

Petroleum Distillates

Kerosene
Mineral seal oil
Mineral spirits
Naphtha high boiling
Spindle oil
Stoddard solvent
Summer black oil

Other Toxic Substances

Antimony chloride
Caustic alkali
Cellosolve
Isopropyl and butyl alcohols
Nitrobenzene
Oxalic acid
Turpentine

Paint Solvents and Related Products

Paint Brush Cleaners and Preservatives

Acetone
Caustic alkalis
Cresols and higher phenols
Dipentine
Methanol
Naphthalene
Sodium chromate
Toluol
Turpentine

Paints Putty Varnishes

Arsenic
Chromium
Iron
Lead
Titanium
Zinc

Removers of Paint Wax Lacquers Grease Spots

Amyl acetate
Alcohols—amyl butyl ethyl
Amylene dichloride
Benzene
Butyl acetate
Carbon tetrachloride
Caustic alkalis
Ethyl acetate
Ethylene dichloride
Kerosene
Methyl alcohol
Methylene chloride
Toluene

Cleaning Polishing and Bleaching Agents

Dry Cleaning Fluids

Acetone
Amyl acetate
Benzene
Carbon tetrachloride
Ethylene dichloride
Kerosene
Methyl alcohol
Naphtha heavy petroleum
Petroleum distillates
Stoddard solvent
Toluene
Trichlorethylene

Detergents for Dishware Glassware Laundry

STRONG ALKALINE SOLUTIONS

Sodium hydroxide
Sodium metasilicate
Sodium perborate
Sodium phosphate glass
Tetrasodium phosphate

OTHERS

Ethylene glycol
Sodium hypochlorite

Metal Cleaners and Polishes

STRONG ACIDS & ALKALIS

Ammonia water caustic soda
Hydrochloric acid dilute
Phosphoric acid dilute
Soda ash
Sulfamic acid
Sulfuric acid dilute

OTHERS

Alkyl aryl sulfonate
Oxalic acid
Potassium chlorate
Potassium cyanide
Thiourea

Cosmetic Preparations

Skin Tonics and Lotions
Aluminum salts
Camphor or menthol
Methylated spirits
Zinc phenolsulfonate
Zinc sulfate

Sun Tan Lotions
Denatured alcohol
Methyl salicylate
Para-aminobenzoic acid esters

Hair Preparations
BRILLIANTINES
Industrial methylated spirit (contains wood naphtha)
Kerosene deodorized

PERMANENT WAVE SOLUTIONS
Sodium carbonate
Sodium sulfite
Thioglycollic acid salts

NEUTRALIZERS
Acetic acid
Potassium bromate
Sodium hexametaphosphate
Sodium perborate

LACQUERS
Denatured alcohol
Shellac resin

HAIR DYES TINTS COLORINGS
Ammonium hydroxide
Ammonium nitrate
Metallic dyes
Paraphenylenediamine
Pyrogallol
Sodium hypochlorite sol

SHAMPOOS
Denatured alcohol
Sodium hexametaphosphate

HAIR LOTIONS
Beta-naphthol
Cantharidin
Denatured ethyl alcohol
Glacial acetic acid
Industrial methylated spirit
Isopropyl alcohol
Pilocarpine
Salicylic acid
Tertiary butyl alcohol

DEPILATORIES
Calcium thioglycollate
Soluble sulfides

Nail Preparations
ORGANIC SOLVENTS
Alcohols—ethyl isopropyl n butyl
Esters
Ketones

LACQUERS, PLASTICIZERS RESINS
Dibutyl phthalate
Nitrocellulose
Sulfonamide resins

CUTICLE REMOVERS
Dilute caustic alkalis

Other Types of Household Products and Chemicals

Antifreeze Carburetor Cleaners
Chlorinated benzene
Alcohols—denatured
Isopropyl methyl
Ethylene glycol

Deodorizing Tablets
Formaldehyde

Anti Rust Products
Ammonium sulfide
Hydrofluoric acid
Naphtha
Oxalic acid

Leather Preservatives Polishes and Dyes
Benzene
Methanol
Neat foot oil
Naphtha
Toluol

Shoe Cleaners and Polishes
Nitrobenzene
Shellac
Titanium dioxide
Turpentine

Inks
Iron gallate
Phenol
Silver nitrate
Soda ash

Jewelry and Watch Cleaners and Cements
Ammonia
Isopropyl alcohol
Nitrocellulose in ketone
Petroleum solvent

Fire Extinguishing Fluids
Carbon tetrachloride
Naphtha

Typewriter Cleaner
Cellosolve
Methanol

Laundry Blue
Prussian blue
Oxalic acid

Bug Adhesives
Latex
Sulfur
Synthetic rubber
Zinc oxide

Plastic Menders Glues
Cellulose acetate
Ethylene dichloride
Formaldehyde
Nitrocellulose

Wax Crayons
Para red

Christmas Bulb Fluid
Methylene chloride

This list of household agents is taken from an adaptation by *American Druggist* of a compilation by Professor Morton J. Rodman of Rutgers University. The authors thank *American Druggist* and Professor Rodman for permission to republish.

separately the stomach and contents intestines and contents, blood liver kidneys and portions of other organs Each specimen should be placed in a clean glass jar without antiseptic and closed hermetically with a lid and sealing wax The containers should be taken or sent to a competent toxicologist for examination and analysis In addition the physician should make a detailed written record of all observations pertaining to the case

The chemical and biologic examination of organs and material are highly technical procedures and must be made by a qualified toxicologist However the physician should be familiar with the toxicologic tests and procedures because he may be called to give expert testimony on the witness stand

5

Local Anesthetic Drug Reactions

ALL PHYSICIANS have many occasions to use local anesthetic drugs in topical applications regional nerve blocks or simple local infiltration For this reason this topic is discussed separately Any physician who uses these drugs is very likely to encounter a reaction to them and the reaction is often acute in onset many times of major proportions and not infrequently fatal Patients are occasionally needlessly lost because the physician is unaware of the proper procedures and the order in which they should be applied and is unprepared with the necessary materials to combat the disorder Many patients who have a reaction to these drugs can be saved if handled properly and a death from such a reaction is often a serious reflection on the physician s preparedness for such emergencies

Unfortunately many physicians consider reactions to local anesthetic drugs an allergic phenomenon Some patients do exhibit this sort of reaction to local anesthetic drugs but most of the reactions are on

an entirely different basis When once the latter fact is understood recognition of the reaction and its therapy are more effective

ALLERGIC REACTION

In patients who do manifest an allergic reaction to these drugs the picture is characterized by the production of skin wheals angioneurotic edema hypo tension occasional bronchospasm and the other phe nomena associated with an allergic reaction from any cause It is entirely different from the usual reaction to local anesthetic drugs If a patient gives a history that includes symptoms and signs of the other men tioned it is advisable to avoid use of the inciting drug or one with a similar chemical structure However if a patient gives a history of symptoms typical of the reaction (see the following pages) it is not necessary to avoid repetition of the offending drug The inci- dence of true allergic reactions to procaine and simi lar drugs is extremely low whereas the incidence of the other type of reaction is not low

SENSITIVITY

It is perhaps true that some patients are more sensitive to these drugs than others in the following respect Since as will be pointed out later, the common reaction to these drugs is dependent on the blood

level of the drug some patients probably manifest the reaction with a lower blood level of the drug than do others It may also be that in some patients the blood esterase which has to do in some fashion with the splitting of the local anesthetic drug into its less toxic forms is present in a smaller amount or is less active This situation could result in slower detoxification of the drug and the development of a high blood level in the presence of lower concentrations and lower total amounts of the anesthetic agent In these patients it is advisable to exercise caution in the concentration the chemical type of drug and the total amount employed It would be unfortunate and unnecessary to deprive these sensitive patients of the advantages of the local anesthetic drugs and use some other less desirable anesthetic agent and technic when with care and close attention to details the drugs could be used satisfactorily and safely

USUAL REACTION

The usual reaction to procaine or similar drugs is due to the absorption of a sufficient amount of the drug into the blood stream to cause symptoms and signs of varying degrees in direct proportion to the blood level and the rapidity of its attainment This is a fundamental principle which if kept in mind will facilitate the prophylaxis recognition and treatment of these reactions

It must be remembered that absorption of these drugs is increased in areas in which the circulation is abundant Examples of such areas are the upper respiratory passages the urethra and the caudal canal which contains a large venous plexus A good general rule for prophylaxis is to avoid whenever possible, the use of high concentrations or large amounts of these drugs in areas in which absorption into the blood stream will be rapid

The reactions to procaine are duplicated by the other local anesthetic drugs Development of a reaction depends entirely on the rapidity with which the toxic blood concentration is reached

An immediate reaction is due to accidental intravenous injection of the analgesic solution or to absorption of a large quantity in a very short time The reaction appears within a few seconds after the injection or topical application of the drug and is characterized by abrupt and complete deterioration of circulation and respiration The patient usually is dead within a minute or two

Most reactions occur 5 10 minutes after injection or topical application If a reaction has not developed within 30 minutes of injection or topical application it is not likely to develop at all The symptoms and signs appear with varying degrees of rapidity and *severity depending on the rate of absorption* and the quantity absorbed

The patient becomes somnolent and the somnolence gradually develops into coma A few patients have enough insight to realize that all is not quite











Fig 29 —Signs of cocaine or similar drug reaction

right and in the earliest phases of the reaction describe themselves as being uncomfortable and somewhat ap prehensive Another small group of patients will be come euphoric and elated This reaction occurs most frequently after the application of cocaine It is always good practice to maintain contact with the patient during the induction of regional infiltration or topical analgesia By so doing one can ascertain the patient s mental state and detect premonitory signs of a reaction If a patient who has been complaining about the insertion of needles or the topical application of a drug abruptly becomes less voluble and apparently more co operative one should not breathe a sigh of relief but rather become apprehensive about the state of the patient

When somnolence or other early signs develop one should palpate the pulse It may be of good quality and approximately the same rate as it was prior to injection or it may be of poor quality and somewhat slower than normal Tachycardia is not the usual finding If the pulse is poor in quality one should check the blood pressure It will be lower than normal if the reaction is progressing Hypotension may be come extreme Associated with the hypotension will be pallor, perspiration clammy skin and syncope Accompanying the development of hypotension will be respiratory irregularities characterized by shallow breathing sighing periods of apnea and occasional dyspnea Twitching about the face and in the fingers may appear and may ultimately develop into a full blown generalized convulsion Respiratory and circula

CORRECT TREATMENT

I GIVE OXYGEN

A. PROVIDE ADEQUATE AIRWAY
B. SUPPLY OXYGEN BY BAG AND MASK.
C. ARTIFICIAL RESPIRATION BY SQUEEZING BAG REGULARLY MASK TIGHT ON FACE
BE SURE CHEST EXPANDS WHEN BAG IS PRESSED.



II ELEVATE BLOOD PRESSURE

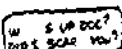


GIVE EPHEDRINE IN 5 MINIM (15 MG) DOSES INTRAVENOUSLY REPEAT UNTIL B.P. RETURNS TO NORMAL

III STOP CONVULSIONS



GIVE PENTOTHAL INTRAVENOUSLY UNTIL CONVULSIONS STOP (A FEW CC. OF 2½% SOLUTION USUALLY ENOUGH 3 5 CC) REPEAT IF CONVULSIONS RECUR



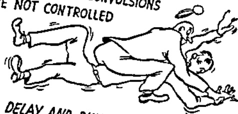




CORRECT TREATMENT WITHOUT DELAY WILL PREVENT A NEEDLESS DEATH!

Fig 30 —Correct treatment of cocaine or similar drug reaction

Fig 31 —Incorrect treatment of cocaine or similar drug reaction



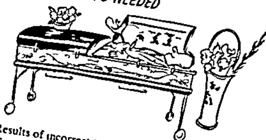


FIG 32 —Results of incorrect treatment of cocaine and similar drug reactions

tory failure succeeds the convulsions if the latter are not controlled

The foregoing symptoms and signs comprise the complete reaction Minor degrees of this major reaction may occur depending on the blood level of the drug

Patients in whom only the mental changes develop require no therapy The injection or topical application of the drug should of course be discontinued One should however be alert to progression of symp-

toms and signs and have material ready for treatment if the reaction becomes more severe

The hypotension is primary shock and must be treated with a vasopressor drug The drug is given preferably intravenously to insure prompt and effective response The vasopressor drug should be administered in repeated doses (every 3 to 5 minutes) until satisfactory blood pressure is obtained In severe states of hypotension massive total doses of vasopressor drugs may be needed to restore and stabilize the blood pressure It is extremely important to elevate the pressure because persisting low tensions may cause enough tissue hypoxia to produce permanent damage

Oxygen therapy should be given when hypotension is present This helps to minimize the danger of anoxia during the period of hypotension and respiratory irregularities A mask and bag are satisfactory particularly if assistance to pulmonary ventilation is required Endotracheal catheterization is beneficial in the presence of severe reactions

The respiratory irregularities are not treated specifically although oxygen and artificial respiration are given if needed

Twitchings and convulsions are controlled by the intravenous administration of a soluble barbiturate Enough barbiturate must be given to stop the convulsion This amount may stop respiration also but artificial respiration can be done easily and it is mandatory that the convulsions be stopped The barbiturate may be repeated until there is no indication that convulsions will recur on discontinuance of the

drug Severe convulsions may be controlled with as little as 0 15 Gm of thiopental sodium or thioamylal sodium

It is important to realize that all of the means for resuscitation outlined must be immediately at hand whenever one contemplates using regional infiltration or topical analgesia Prompt and courageous therapy can prevent fatal termination of the reaction in a large majority of patients whereas minimal delays in instituting therapy and in using it thoroughly will unnecessarily increase the incidence of fatal outcome

Minimal equipment that should be instantly available whenever one intends to use analgesia of the types under discussion includes a tank of oxygen a reducing valve a mask and rebreathing bag a pharyngeal airway a vasopressor drug and a soluble barbiturate Where regional infiltration or particularly topical analgesia with cocaine *or tetracaine is used frequently it is advisable* to have the soluble barbiturate *in solution in a syringe ready for instant injection* This last precaution is a life saving factor The equipment mentioned may not be used for years but if it is used only once and a life is saved because it was there for immediate use it will prove its efficiency *and* value *It* should be present and *immediately* available in every physician s office

Epinephrine in Anesthetic Solutions

The foregoing discussion raises the question of the advisability of combining epinephrine with anesthetic solutions It is recognized that epinephrine permits the

use of lower concentrations and lengthens action presumably by delaying absorption All of these factors tend to prevent accumulation of sufficient quantities in the blood stream to cause reactions The use of epinephrine in concentrations of 1 100 000 or 1 200 000 is effective and probably beneficial It must be remembered that epinephrine in higher concentrations may result in undesirable degrees of ischemia and unpleasant reactions The ischemia may be sufficient to precipitate gangrene Secondary vasodilation may also occur with consequent hemorrhage in the postoperative period Reactions to epinephrine are characterized by tachycardia palpitation apprehension and hypertension Epinephrine reactions should be differentiated from procaine or similar drug reactions when the drugs are used together The hypertension makes it unwise to use epinephrine in appreciable quantities in patients with hypertension Treatment of a reaction to epinephrine is symptomatic Oxygen inhalation supports the heart through a period of overactivity and sedatives allay apprehension The reactions are seldom fatal and do not last long

It is possible to prolong the action of the anesthetic solution through the use of Cobefrin with it This drug does not have the cardiac and circulatory effects that epinephrine does and is a satisfactory substitute It is used in concentrations of 1 5 000 to 1 40 000

6

Head Injuries

1 Preliminary examination first aid measures as required
2 Combat shock if present
3 Examine thoroughly record findings as early as possible
4 Transport the patient if necessary
5 Institute conservative measures
6 Re-examine at frequent intervals record observations
7 Institute surgical measures if and when indicated
8 Resume conservative measures including repeated examinations

1 PRELIMINARY EXAMINATION

Of necessity the preliminary examination must be brief The aim is rapid appraisal of data gained by inspection palpation and manipulation Within the first few moments the state of the patient s vital functions can be taken in Pallor rubor cyanosis and inordinate sweating are at once obvious The skin temperature respiration and the rate regularity and

force of the pulse can be estimated by rapid palpation During these observations, the physician should formulate a rough notion as to the existing state of consciousness by evaluating the promptness vigor, co-ordination and adaptiveness of the patient s responses to such happenings in the environment as verbal directions to him a puff of air blown against the eyelids and pinching of the pectoral tendons Rambling delirium confusion maniacal excitement resistiveness and combativeness, retching vomiting and incontinence are self-evident Likewise asymmetries of volitional and emotional movements of the face and limbs and unusual postures of the head jaw trunk and extremities will be apparent

The patient should be carefully inspected for evidences of active or arrested hemorrhage in the aural buccal and nasal orifices Simultaneously nuchal rigidity is tested for Lacerations of the scalp and integument and gross evidences of fractures and dislocations of the long bones spine ribs and pelvis are next sought The possibility should not be overlooked that hemorrhage may be concealed e g, in the retroperitoneal space thoracic cavity and Hunter s canal at the site of fracture of the femoral shaft

First Aid Measures

In the head injured as in others with trauma 2 emergency conditions demand prompt measures impending asphyxia and hemorrhage The former is usually the more urgent The neural and humoral

dysfunctions set into motion by partial even subclinical degrees of asphyxia rapidly lead to irreversible tissue damage and not infrequently to death The treatment of asphyxia is covered in Chapters 1 and 2

Asphyxia —The hypoxic state is most acutely felt by the cerebral cortical cells and next in order by the nuclear structures of the basal ganglions cerebellum diencephalon and brain stem Without sufficient oxygen the cerebral tissues are unable to utilize glucose and the resulting intracellular respiratory and metabolic embarrassment is soon followed by the development of spreading edema The edema by compromising the caliber of capillaries precapillaries and venules and interposing a further physicochemical barrier to cellular respiration fosters the development of even more severe cerebral hypoxia and edema The vicious circle is completed when the respiratory and circulatory neural mechanisms in the frontal lobes hypothalamus midbrain and medulla become involved In such circumstances pulmonary edema rapidly supervenes and the patient enters upon a preagonal state from which recovery may not reasonably be anticipated

The neural respiratory complex appears to be far more susceptible than the cardiovascular mechanism to equal degrees of physiologic insult whether of traumatic or other character In fact it has been demonstrated experimentally that the otherwise healthy cardiovascular mechanism can be relied on to perform its functions with machine like precision provided a proper respiratory exchance is maintained When however hypoxia is permitted to develop the aforemen

tioned evidences of circulatory dysfunction rapidly supervene These signs may be erroneously attributed to increased intracranial pressure As the oxygen want grows the blood and pulse pressures diminish and the pulse rate becomes accelerated In such a case efforts to support the heart and circulation with digitalis caffeine and ephedrine are obviously misdirected and usually fail

POSITION —The usual practice of lowering the head of the unconscious patient is contraindicated in the presence of craniocerebral trauma Instead, elevate the head so that it rests at a slightly higher level than that of the heart This positioning can be accomplished by elevating the bed on shock blocks or using cushions folded overcoat or similar convenience

Hemorrhage —Active hemorrhage is the second condition requiring prompt attention If the wound is on the scalp the neighboring skin should be shaved The wound is then irrigated with an innocuous antiseptic such as Zephiran chloride® 1 2 000 The means adopted for control of hemorrhage vary with the site of laceration and with whether an artery or a vein is involved Digital compression and application of a tourniquet or of hemostatic clamps are the speediest measures Direct ligation of the bleeding vessel and closure by the placement of on end mattress sutures in the surrounding tissues are equally useful although more time-consuming When the hemorrhage has been controlled a snug dry gauze dressing should be applied

2 SHOCK

The treatment of shock in the patient with a head injury takes precedence over that of any other condition Transportation and excessive manipulation of the patient are to be avoided This rule occasionally must be violated especially when cold wind rain rough ground or heat of the sun interferes with the proper conduct of therapy In such circumstances the movement should be planned and executed with dispatch care being exercised to avoid jarring and gross postural displacements

Measures for combating shock are described on page 107 However because of some long standing concepts regarding the management of shock 3 exceptions to older concepts are emphasized They are directly applicable to the treatment of shock complicating craniocerebral trauma The undesirability of lowering the head has already been mentioned The reason for this is that such posture favors intracranial venous congestion and hence the development of cerebral edema A compromise in the form of a horizontal posture is indicated As soon as the patient has been brought out of shock and the vital signs have been stabilized the head should be elevated to 15 20 degrees above the horizontal

The second exception relates to the routine administration of morphine and other opium derivatives to the patient in shock This should be avoided in the head injured for several reasons the chief of which

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is that stuporous and unresponsive patients require no relief from pain anxiety and restiveness The principle seems axiomatic, but it is far more frequently violated than honored Other cogent reasons for the interdiction of narcotics are the undesirable depression of respiration slowing of motor and secretory functions of the gastrointestinal tract and antidiuretic action Morphine moreover produces pinpoint pupils a condition which may seriously mask or retard the development of one of the most suggestive of cerebral localizing signs namely unilateral pupillary dilatation Finally the administration of morphine is commonly followed by a rise of cerebrospinal fluid pressure of between 50 and 120 mm of water Such an increment superimposed on established intracranial hypertension due to cerebral edema or hemorrhage might easily turn the tide against the patient

The third exception relates to the use of concentrated (25 50 per cent) glucose solutions and of strongly hypertonic electrolyte solutions in general With brain trauma these agents frequently prove unpredictable and sometimes actually harmful At the site of cerebral contusion laceration and/or edema there is vascular discontinuity and the hypertonic solution simply fails to reach and perfuse the region where its dehydrating action is most desired Moreover it may spill through the damaged vessels infiltrate the local tissues and provoke untoward electrolytic effects

Shock ascribable to craniocerebral trauma is as a matter of actual record, rather uncommonly encoun

tered by the physician This is not meant to imply that shock is rarely produced by trauma to the head for without doubt it may be severe enough to kill the patient in a few minutes The statement is meant rather to emphasize the point that *if* the initial shock alone is not severe enough to kill, recovery tends to be so rapid that by the time the patient is seen he is usually out of shock or at least rallying effectively As a consequence of an unselected group of patients with acute head injuries only 6 8 per cent present shock or soon develop it And most of these prove to have extracerebral causes for the shock damage to the lungs liver, spleen adrenals kidney and bladder, fractures of the ribs long bones pelvis and vertebrae exsanguination from lacerations of large vessels of the extremities scalp and viscera exhaustion and malnutrition burns exposure to excessive heat or cold and severe intoxication from alcohol and other drugs In only about 0 5 per cent of the unselected group does it appear warrantable to ascribe the shock to the cerebral trauma per se

The physician aware of these implications is unlikely to assume that shock in a head injured patient is due to the brain trauma He is more likely to make an extracerebral search for the responsible factor—and to be rewarded by its disclosure

3 THOROUGH EXAMINATION

After all indicated therapeutic measures directed against asphyxia hemorrhage, shock and similarly

urgent conditions have been instituted, a more thorough general and neurologic survey is possible

During the general physical examination of the stuporous patient one precaution must be exercised when it is necessary to turn him e g from the supine to the lateral or prone position the movement should be carried out *gently and in log fashion* Not a few patients with cerebral trauma have died because of faulty turning technics The head should be passively supported and the sagittal planes of head shoulders and pelvis kept in strict alignment to prevent injury to the spinal cord lungs and other viscera from undetected damage to adjacent bony structures To turn the average adult under proper control usually requires the co-ordinated efforts of the physician and at least two assistants—one managing the head a second the shoulders and upper trunk and a third the lower trunk and limbs

The neurologic examination of the head injured subject is likely to be incomplete as compared with ideal standards particularly if he is confused aphasic unco-operative hypomaniacal or unconscious In such circumstances one is obliged to resort to the observational technics regularly employed by the veterinarian He should endeavor to discover disparities in the corneal nasal abdominal and plantar reflexes and in the biceps patellar and Achilles jerks Gross changes that have developed since the initial examination are often apparent in speech functions, pupillary responses extraocular and facial movements coordination and motor power of the extremities A

satisfactory sensory examination is rarely possible However the demonstration of differences in defense responses to stimuli delivered to corresponding parts of the right and left sides of the body may afford a crude measure of the patient s sensory status

The optic fundi should be routinely examined for evidences of retinal hemorrhages increased ratio of vein—artery caliber and early signs of papilledema Mydriatics should be avoided whenever possible be cause they mask the development of pupillary in equality Except in overwhelmingly severe injuries the ophthalmoscope rarely discloses unusual findings with in the first 48 hours

Records —A written record of the significant posi tive and negative findings disclosed by the general and neurologic examinations should be made at the con clusion of each survey Such a record provides a base line to which all later clinical findings may be referred and helps the physician to judge whether the patient is holding or losing ground gaining or vacillating

Reading of the blood pressure pulse rate re spiratory rate and rectal temperature should be recorded every 15 minutes for the first 3 hours and for uch time thereafter as clinically significant variations con tinue The interval between successive readings may be later increased depending on whether or not the vital signs give evidence of stabilizing at near normal levels

Evaluation of vital signs —Stabilization of vital signs even at levels somewhat above normal gener ally renders the prognosis more favorable however

this criterion cannot be implicitly trusted As a general principle instability of vital signs is more a sensitive and reliable indicator of ensuing serious trouble than the patterns of signs and symptoms related to such classic syndromes as epidural clot and increased intracranial pressure Bradycardia of 50 or 60 beats per minute is not uncommon even after mild head trauma and of itself need not arouse much anxiety But blood pressure readings above $^{160}/_{100}$ and under $^{8}\ _{0}$ mm Hg and/or appreciable fluctuations of pulse and blood pressure even if within these ranges are grave signs Fluctuations in the rate depth and rhythm of respiration often presage the development of Cheyne Stokes breathing If as usually happens respiratory fluctuations are associated with appreciable variations in the force and rate of pulse there is sufficient evidence to justify surgical inquiry

Deepening stupor and progressive development of pupillary disparities conjugate deviations of the eyeballs, inco ordination of extraocular movements aphasia paralysis spasticity twitchings and convulsive phenomena constitute grave signs Their development like the instabilities of vital signs indicates consideration of radical investigative procedures in the form of parietal burr openings direct inspection of the brain and if indicated ventriculography

4 TRANSPORTATION

Unless in shock the head injured patient tolerates transportation well for distances upward of 100 200

miles as amply confirmed by neurosurgical experience during World War II It is far better to transport the patient to a hospital where facilities for sustained observation medical and nursing care and prompt surgical intervention are available than to retain him at the first convenient stop under the misapprehension that to move him will jeopardize his condition

Once it has been decided to move the patient he should be moved to and from the litter in log fashion and unless there are special reasons to the contrary transported prone with the body turned slightly to the side One arm is left at the side of the body and the other brought forward so that the head can rest on it

5 CONSERVATIVE MEASURES

When he arrives at his destination the patient should be placed in a posture which facilitates easy respiration Orders should be left for him to be turned every two hours to prevent the development of pulmonary hypostasis and decubitus ulcers Prophylactic antibiotic and sulfonamide therapy may be instituted if development of infection at the site of trauma seems likely With compound fractures of the vault and jaw or of the base of the skull communicating with the middle ear paranasal sinuses or pharynx the use of these agents in large doses is mandatory Tetanus and gas gangrene antiserums may be indicated especially when the wounds provide anaerobic conditions for spore bearers Needless to say the usual pre

is readily detected by percussion of the lower part of the abdomen Occasionally gratifying amelioration of restiveness can be accomplished by catheterization

Orificial Bleeding

Bleeding from the nose or ear whether with or without cerebrospinal fluid need not excite undue alarm It usually ceases spontaneously in a few hours or days, and surgical intervention is rarely necessary during the acute phase The involved orifice may be gently washed or swabbed out as occasion demands but packing it instilling medicaments and irrigating it with antiseptic solutions are strongly contraindicated because of danger of pyogenic meningitis The posture should facilitate gravitational drainage If the patient is rational and able to co operate he should be urged to suppress coughing and sneezing Penicillin and/or sulfonamides should be given prophylactically

Fever

One of the more conspicuous clinical features of severe brain injury is the development of anhidrotic fever It is important to recognize that *in and of itself hyperthermia may kill the patient* In the absence of sweating the dissipation of body heat depends chiefly on accelerated respiration and the physical mechanisms of convection conduction and radiation In hot weather (and to a lesser degree at ordinary room temperatures) these mechanisms often prove incom

petent since they require a relatively steep gradient between the body temperature and that of the environment. Every precaution must therefore be taken from the outset to keep hyperthermia under control. If possible the room temperature should be maintained at 55-60 F. Rectal temperature should be taken every 20-30 minutes. If the successive readings are near normal, remain stabilized or fall in the direction of normal, temporizing is permissible and the interval between temperature readings may be increased to 1 or 2 hours.

TREATMENT. Whenever the rectal temperature exceeds 102.5 F. active measures must be adopted at once to combat fever: the patient should be completely stripped of his covers and bed clothes, an electric fan played on his exposed body and continuous sponging of limbs and trunk with a wash cloth moistened in a tepid 50-50 solution of alcohol and water should be instituted. *These measures must be continued until the rectal temperature falls below 101.5 F. and are to be re-instituted whenever the critical level of 102.5 F. is again exceeded.* If after an hour or so of continuous sponging or fanning the temperature fails to recede and certainly if it continues to rise, the colonic instillation of cool water must be instituted. A necessary preliminary is the simultaneous determination of rectal and axillary temperatures to ascertain an axillary equivalent of the rectal temperature. A rectal tube is now inserted some 18 in. into the colon and 3.5 L. of cool tap

(1) the determination of whether or not blood contaminates the cerebrospinal fluid (and is so, approximately how much), and (2) the measurement of cerebrospinal fluid pressure Both questions can be answered with the loss of but 1-2 cc of fluid The removal of more fluid is unnecessary for diagnostic purposes

The information afforded by early lumbar puncture offers a basis for evaluating subsequent clinical developments Thus if the cerebrospinal fluid at the time of the initial tap is water-clear and under normal pressure it is unlikely that cerebral contusions or lacerations with attendant subarachnoid subdural intracerebral or intraventricular hemorrhages are present Should the patient then lose ground i e, develop instabilities of vital signs and/or aberrant neurologic findings a suspicion of an epidural clot would be warranted particularly if lumbar puncture now reveals clear fluid but under increased pressure In these circumstances surgical inquiry is imperative If however the cerebrospinal fluid at first is contaminated with blood and pressure is normal or nearly normal (up 250 mm of water), the inference that cerebral contusion subpial and subarachnoid hemorrhages exist is justified This should prepare the physician for possible development of cerebral edema and increase in cerebrospinal fluid pressure and if the patient loses a little ground the necessity for immediate surgical exploration will be somewhat less urgent than in the former case

We are not convinced as some colleagues are that the severity of cerebral damage can often be ex pressed in terms of the pressure of cerebrospinal fluid or that quantitative measure of the pressure is at any given moment a valid criterion of the therapy called for The data obtainable by lumbar puncture do not permit unequivocal conclusions However when evaluated in the light of other clinical data they aid in arriving at a defensible diagnosis and for this reason the routine use of diagnostic lumbar puncture seems justified

To obtain reliable information from lumbar puncture the manometric examination must be carried out with the patient in the lateral horizontal posture relaxed and with no restrictions to respiration The *normal pressure range of cerebrospinal fluid lies between* 6 and 12 mm Hg (80 and 180 mm of water) In the head injured patient the Queckenstedt test should not be carried out Not only is it incapable of yielding information as to the presence of intracranial lesions but it may precipitate hemorrhage from dam aged intracranial vessels

The value of lumbar puncture as a therapeutic procedure is a matter of much controversy Some au thors contend that intracranial hypertension and cere bral edema can be successfully controlled in most cases by repeated spinal taps withdrawing quantities of fluid ranging from 30 to 60 cc up to complete emptying They contend that blood extravasated into the subarachnoid space acts as a meningeal irritant and a deterrent to the escape of cerebrospinal fluid

into the venous system and that it can be largely eliminated as a contaminant by repeated drainage of the fluid Other students deny such virtues to repeated drainage They express concern over the potential dangers of therapeutic lumbar puncture, contending that the reduction of intracranial pressure may permit the reopening of recently occluded vascular channels that it may result in herniation of the temporal lobe through the tentorium cerebelli or of the cerebellar tonsils and medulla through the foramen magnum and that it usually excites a compensatory overproduction of cerebrospinal fluid, such that in 2 to 3 hours the intracranial pressure may be appreciably higher than it would otherwise have been They further hold that gross hemorrhagic lesions whether epidural subdural or intracerebral capable of increasing intracranial pressure cannot be favorably influenced by withdrawal of fluid In the presence of such lesions any salutary effect of lumbar drainage is short lived and likely to be beset with the dangers that attend false optimism They insist that the patient s margin of safety is appreciably reduced after therapeutic tap because such nondisplaceable elements as cerebral edema and collections of blood are now enabled to take up space previously occupied by the more displaceable cerebrospinal fluid

The relative merits of these arguments cannot be evaluated here There appears, however, little objection to the occasional use of therapeutic lumbar puncture as a stop gap measure to permit one or more hours delay before surgical intervention

HYDRATION AND FEEDING

On the whole vigorous dehydration in the early management of head trauma appears to have little merit One may safely order magnesium sulfate 45 cc or 1½ oz of saturated solution by mouth once or twice daily and/or caffeine sodiobenzoate 500 mg (7½ gr) intramuscularly 4 to 6 times daily But with rare exceptions the daily fluid intake should exceed 1 800 2 000 cc and in the presence of hyper thermia restiveness delirium tremens, etc well over 3 500 cc is required The unconscious and restive patient should not go more than 24-48 hours on mere parenterally administered fluids If at the end of this period he is still unable to take nourishment by mouth a Levin tube should be passed via the nose and the fol lowing schedule of hourly feedings instituted

1st hour glucose water 120 ml (4 oz)
2d hour sweetened fruit juices 120 ml (4 oz)
3d hour eggnog, 120 ml (4 oz)

This sequence of feedings is repeated throughout the 24 hours Three grams of table salt most convenient ly administered in suitable fractions with the eggnog should be added to the daily diet The general superior ity of the method of administering nutritive and medic inal agents by Levin tube over intravenous and sub cutaneous methods is obvious

Experience indicates that the combination of a tracheotomy or endotracheal tube and a Levin tube invites development of a tracheoesophageal fistula This danger may be reduced by substituting a slender

polyethylene tube of 2 3 mm diameter for the Levin tube

Convulsions

Although highly dramatic convulsions do not in themselves necessarily constitute a serious prognostic sign This is particularly true in connection with isolated *(as opposed to repeated)* seizures *Children are* likely to exhibit one or several seizures following relatively minor head injuries Here again no great anxiety need be felt

Once under way a convulsion cannot be readily aborted nor need it be *The only procedures immediately necessary when a convulsion supervenes are those concerned with protecting the patient from asphyxia and self injury* There is no need to pry the jaws apart to gag a mouth already clenched in spasm serious injuries to the lips tongue and teeth may result from such efforts

TREATMENT Repeated convulsive seizures (status epilepticus) demand active anticonvulsant measures for the patient may rapidly become exhausted from such attacks The most effective anti convulsive drugs are the barbiturates by mouth vein or muscle the bromides and Dilantin Sodium® by mouth Avertin® by rectum and ether by inhalation (see Chapter 8) An observer should record accurately the sequence of motor events leading up to the convulsive attack Such data often provide a valuable clue to the locus of an epileptogenic firing

point and this in turn may go far in determining the advisability of surgical therapy and the site of ex posure of the brain

General Care

Unconscious patients breathing for hours through the relaxed mouth are subject to parotitis Therefore it is important to cleanse the mouth of dried saliva every 3 to 4 hours A useful solution for this purpose consists of equal parts of glycerin and lemon juice A simple humidifier in the form of a water moistened gauze compress laid across to external air passages is helpful

Although urinary retention is occasionally serious in stuporous and disturbed subjects incontinence is much more common It is essential if the develop ment of decubitus ulcers it to be averted that bed linen be changed as soon an soiling occurs If desired and indwelling catheter may be passed into the blad der This may be closed with a Hoffman clamp or hemostat and the urine released every four to six hours A small quantity of antiseptic solution such as Zephiran Chloride® 1 5 000 should be instilled after each evacuation In patients requiring more prolonged care intermittent or tidal irrigation is more practical than the method of intermittent release In either case the urine should be examined for evidence of cystitis and the catheter should be renewed every few days

During the first 2 to 3 days after head injury little attention need be given the bowels Incontinence

of stool is not nearly as common as urinary incontinence When magnesium sulfate is employed as a dehydrating agent incontinence of stool is likely to appear Otherwise unless relieved by enemas the head injured patient may go for days without a bowel movement Fecal impactions are likely to develop in unconscious patients and should be sought whenever abdominal distention appears and when inordinate constipation is suspected

6 RE EXAMINATION AT FREQUENT INTERVALS

The foregoing paragraphs point up the major considerations demanding attention in the early conservative management of the head injured patient Provided no indications for surgery arise, the patient may be carried on this regimen for weeks and months without impairment of his general health It is essential however, that general physical and neurologic examinations be repeated at intervals indicated by the objective changes There can of course, be no hard and fast rule for this It is urged again however that the physician continue to record all positive and negative findings and to compare them with one another and with the first established baseline

7 SURGERY

If during conservative management the patient begins to exhibit (*a*) evidence of increasing stupor (*b*) persistent trends of the vital signs away from

normal ranges and/or frequent fluctuations within normal and near normal ranges or (c) progressive neurologic dysfunction one should seriously suspect the presence of a surgically amenable lesion The physician must be constantly alert to the necessity of deciding whether and when conservative measures should be supplemented by surgery This is a difficult task and involves large responsibility

The lesions that commonly demand surgery in the early days after head trauma are (1) compound fractures of the vault with or without depression in driven bony fragments foreign bodies pulpifica tion devitalization and/or extrusion of brain tissue (2) simple depressed fractures of the vault such that the outer table of the depressed portion is at or deeper than the inner table of the adjacent vault (3) epidural hemorrhage (4) subdural hemorrhage or hemohy groma and (5) gross intracerebral and/or intraven tricular clots The first two because they are usually immediately apparent or at least strongly suspected from external signs alone rarely present diagnostic difficulties They are moreover readily confirmable by x ray

Of the three other conditions *epidural hemorrhage* is by far the most urgent Clinically it is often diffi cult if not impossible to differentiate epidural sub dural and gross intracerebral hemorrhages and cere bral edema When therefore the presence of *any* of these lesions is suspected the following empiric di agnostic procedures should be undertaken promptly

The first step consists in placing burr openings over both parietal regions In stuporous and uncon

scious patients this can be carried out under local anesthesia Disturbed patients require general anes thesia in which case it is highly desirable to pass an endotracheal tube to guarantee a free airway

Approximately 80 per cent of epidural and sub dural clots are disclosed through burr openings in the parietal bosses Most epidural hemorrhages consist of solid clot If such a lesion is encountered a small temporoparietal bone flap is required to permit its complete evacuation and ligation or fulguration of the lacerated middle meningeal artery Subdural collec tions on the other hand are likely to consist partly of free fluid and partly of solid clot The proportion of fluid and solid components varies considerably from patient to patient although generally there is more solid clot in the recent than in the subacute and chronic case In any event a fluid collection amount ing to 20 cc or more justifies repeated irrigation of the subdural space with normal saline through a soft cath eter until the return flow is clear Even if a portion of the subdural hemorrhage consisting of solid colt re- mains evacuation of the fluid may give sufficient relief to warrant deferment of further surgery until the pa tient s condition is stabilized The solid portion may be attacked later through a small bone flap In deal ing with the severely injured patient the prudent operator limits surgical procedures to those which afford effective even if only temporary relief The overzealous completion of a surgical procedure may overtax the patient with reduced reserve

If an epidural or subdural hemorrhage is not dis closed through the parietal burr holes one is not warranted in concluding that these or other surgi cally amenable lesions are not present Ventriculogra phy is now indicated Such a lesion almost always betrays its presence by displacing or distorting the ventricular system If then the ventriculograms ap pear essentially normal one may assume that further surgery cannot be of help and conservative measures may be reinstituted However a ventricular shift or distortion calls for reflection of a bone flap and ex ploration for an epidural subdural intracerebral and/ or intraventricular clot Any such lesion plus pulpified edematous and devitalized brain tissue hould be evacuated If none is demonstrable one may infer that the ventricular distortion was produced by focal edema of the brain In that case the brain will be swollen and the bone flap should be sacrificed even though nonspecific decompressive procedures of this type can rarely be relied on to serve their intended role

8 RESUMPTION OF CONSERVATIVE MEASU ES

Treatment obviously does not end with the com pletion of definitive surgical measures The conserva tive regimen followed before operation must be resumed and should be continued as long as the pa tient s condition requires Controls should be relaxed slowly and with unremitting vigilance for signs that indicate the necessity for their reinstitution

SUMMARY

The early management of the head injured patient is charged with difficulties engendered largely by two circumstances (1) neurologic signs and symptoms are due to alterations in neurophysiologic mechanisms rather than the initial pathologic processes and (2) multiple rather than unit factor lesions are the rule For these and other reasons the scientifically ideal procedure of establishing a diagnosis and then instituting appropriate therapy frequently must be compromised In actual practice diagnosis and conservative therapy generally proceed in parallel It is not unusual for the former to lag far behind the latter and in many instances particularly when the outcome is favorable a diagnosis may never be verified Fortunately it is not invariably necessary to know how and where a fire started in order to extinguish it

A full appreciation of these circumstances and their implications leads to a readiness to make use of all appropriate diagnostic procedures Therapy can then be altered as new clinical observations are made The early management of the head injured patient can be conveniently outlined somewhat after the manner used by the chemist confronted with a problem in qualitative analysis (Fig 33)

Paradoxical as it may seem a kind of confidence develops hand in hand with the exercise of the principles of procedure based on this brand of agnosticism—a confidence that stems from the realization that by one means or another the appropriate ther

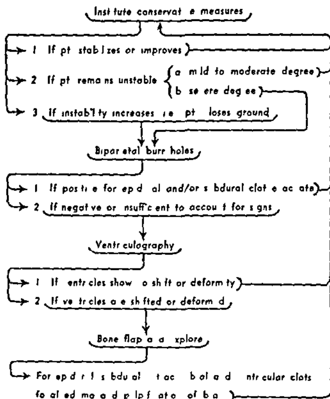


Fig 33.—Determinants of procedure for management of head injuries

apy whether conservative or surgical can and will be made available to each patient, that unnecessary surgical procedures will be held to a minimum and that few patients with surgically amenable lesions will die without having had the benefit of surgery

7

Allergic Disturbances

ALLERGIC reactions often occur very quickly and frequently must be treated as emergencies The most serious of these are bronchial asthma anaphylactic shock from serum or drugs and angioedema of the larynx Less serious but often requiring immediate treatment are such conditions as hay fever, serum sickness and urticaria Some of the abnormalities that occur in these conditions are spasm of the bronchial muscles hypersecretion of the glands capillary dilatation increased permeability of the capillary wall edema of the mucous membrane emotional unrest anxiety and asphyxia They do not occur in equal proportion in all cases and some may be absent in a particular condition The control of allergic disorders of the immediate wheal type is chiefly dependent upon sympathomimetic amines antihistamines adrenocorticals or corticotropin

ASTHMA

In the patient who is inclined to have acute paroxysmal or prolonged attacks of dyspnea the cause ap

pears to be spasm of the bronchial muscles plus edema of the mucous membranes The blocking of the bronchi by thick tenacious mucus also may play a role The onset of an acute attack may be very sudden with rapid appearance of dyspnea and often cyanosis severe enough to suggest acute asphyxia Such severe attacks must be relieved otherwise severe hypoxia and convulsions may occur There are great variations in the severity of the attacks and they may last from a few minutes to several hours or days before the patient has relief

The antihistaminic agents have not been successful in asthma although they may be useful as adjuncts to treatment The acute attacks should be treated by epinephrine or other sympathomimetic amines with epinephrine the choice for severe attacks It is usually administered subcutaneously or given by inhalation as a spray Epinephrine should never be given intravenously unless the patient is extremely resistant to the drug given subcutaneously Experience may show Isuprel Hydrochloride® to be preferable to epinephrine This agent does not have as great a pressor activity as does epinephrine but it is an effective bronchial dilating agent

EPINEPHRINE In children a subcutaneous injection of 0 1 0 3 cc of a 1 1 000 solution is given and repeated at 20 30 minute intervals if necessary *In adults the dose varies from 0 2 to 0 5 cc* repeated at 10 20 minute intervals until relief is afforded Larger doses are to be avoided because they apparently do

not give greater relief and may add to the patient's apprehension and cause acceleration of the pulse rise in blood pressure, throbbing headache tremors and pallor

A suspension of epinephrine base in oil has the advantage of prolonged action thus requiring less frequent injections The initial dose is the same as that for epinephrine hydrochloride and always should be given intramuscularly in the deltoid muscle Reactions such as headache dyspnea chills perspiration tremors and nervousness may occur Injection into the deltoid muscle permits use of a tourniquet intermittently to regulate the absorption of the drug to prevent these symptoms

Inhalation of a spray containing epinephrine is a very convenient method of administration A 1 100 solution is placed in a glass atomizer and sprayed into the back of the mouth during inspiration about 5 minutes is required for action to take place

In a few instances it may be necessary to administer epinephrine intravenously but this should be reserved for patients in whom 1 cc of epinephrine solution 1 1 000 given subcutaneously does not produce symptoms such as tremors For intravenous use 0 1 0 15 cc of epinephrine solution is diluted with 1 cc of saline and given at the rate of about 0 1 cc every 30 seconds It may be followed by a subcutaneous injection to prolong the action Intravenous injections of epinephrine are always dangerous and extreme caution must be exercised when they are given The action is so

rapid that little or nothing can be done to combat severe toxic symptoms if they occur

Reactions and contraindications to epinephrine— The mild untoward effects of epinephrine are anxiety, tenseness restlessness throbbing headache tremor, dizziness respiratory distress and palpitation The administration of large doses subcutaneously or accidentally by vein may have more serious results such as cerebral hemorrhage and cardiac arrhythmias Individuals with hyperthyroidism are particularly susceptible In patients with long standing bronchial asthma who may have degenerative heart disease extreme caution must be used in the intravenous administration of epinephrine Anginal attacks are often induced by epinephrine in patients with angina pectoris

ISUPREL® This drug is a powerful bronchodilator and may be effective in cases resistant to epinephrine It is administered by inhalation from a nebulizer The dose by inhalation should not exceed 0 5 cc of 1 200 concentration at any one treatment The drug causes no significant blood pressure effects but because it does produce tachycardia it should not be used with epinephrine

EPHEDRINE This is not as reliable as epinephrine and in severe attacks is seldom effective however, it may abort an attack in its early stages Its advantages are that it may be given by mouth and the action is more prolonged The usual dose is 15 60 mg (¼ 1 gr) by mouth however larger doses such

as 0 12 0 18 Gm (2 3 gr) may be given without toxic effects Tolerance to ephedrine is quickly gained

AMINOPHYLLINE Aminophylline given in travenously quite effectively relieves bronchial asthma The dose of 0 25 0 5 Gm (4 8 gr) is injected very slowly for rapid administration causes a precipitous fall in blood pressure Children may be given amino phylline suppositories containing 0 12 0 24 Gm (2-4 gr) depending on age but prolonged use should be avoided for serious toxic symptoms may follow

POTASSIUM IODIDE This expectorant should be given early in the treatment of bronchial asthma although its effect may not be noted for 12 to 24 hours Five to 15 drops of the saturated solution of potassium iodide 3 times a day by mouth coupled with hydration of the patient is an effective means of reducing mucous plugging of the bronchioles

CORTICOTROPIN AND ADRENOCORTICALS Many attacks of bronchial asthma are not completely relieved by bronchodilators and expectorants and may persist for days or weeks Corticotropin in 25 mg doses every 6 hours intramuscularly or 25 mg in 500 cc of 5 per cent glucose in water intravenously over a 6 to 8 hour period once every 24 hours will have an anti inflammatory effect on the bronchioles in 6 to 48 hours Prednisone in 15 mg doses every six hours orally is almost as effective as the corticotropin After the attack has subsided the dose of both hormones should be reduced slowly over several days to prevent

the signs of hypoadrenalism In certain patients prednisone may be continued for several weeks or months at maintenance doses of 10 20 mg a day

ANGIOEDEMA AND URTICARIA

In angioedema there is swelling of the skin and subcutaneous tissue of the forehead eyelids lips and throat Sudden edema of the larynx may prove fatal Urticaria or hives is characterized by elevated skin patches about the size of mosquito bites in giant urticaria the lesions are much larger Rather severe itching accompanies the condition The lesions last from a few hours to several days or weeks Epinephrine injections similar to those used in asthma (p 208) are quite effective The antihistaminic agents often give relief when epinephrine fails

In severe prolonged cases corticotropin and prednisone may be useful in suppressing symptoms for short periods Doses are similar to those used in asthma (p 211)

ANTIHISTAMINICS A large number of antihistaminics are on the market and the one to use is a matter of personal choice All appear to be highly effective and selection rests on the individual response Minor side effects are not unusual Gastric upsets sleepiness nervousness sweating fatigue anorexia frequency or urination dry mouth and some numbness of extremities can occur Benadryl® and Pyribenzamine® are more likely to cause sleepiness than is Theph

orin® in fact Thephorin® seems to have fewer untoward effects than either of the others When unpleasant effects become too annoying reduction of dosage may be necessary or another antihistaminic should be used Benadryl® and Pyribenzamine® are usually given in doses of 25 50 mg Chlor trimeton® in 4 mg doses every 6 to 12 hours is an effective antihistaminic with only minimal side reactions Sustained release tablets and capsules of 8 mg and 12 mg doses are available as Chlor trimeton Repetabs® and Teldrin® In very young children 1/4 to 1/2 the adult dose can be tolerated

SERUM DISEASE

Serum disease is an allergic reaction caused by the introduction of a foreign serum The time of onset varies usually being 7 12 days but it may appear sooner if the individual has had serum previously Adenopathy urticaria diffuse erythema itching pyrexia and edema may be present The joints are usually painful and tender Occasionally vomiting diarrhea abdominal cramps and headache accompany the condition

TREATMENT Antihistaminic agents are used and epinephrine is given subcutaneously The pruritus may be partially relieved by calamine lotion and sodium bicarbonate baths Low concentrations of procaine intravenously have been recommended The dose is calculated on the basis of 4 mg per kg (2 mg per lb) given as 0 1 per cent solution in saline over a

20 minute period If minor symptoms of a reaction to the procaine develop (p 170) its administration is slowed If the reaction progresses the drug should be discontinued and the patient treated as outlined on page 174 The injections may be repeated at least twice a day

SERUM SHOCK

This condition may occur in individuals who have had previous injections of horse serum or are spontaneously sensitive to it The onset is immediate and may be fatal in a few minutes Owing to the severity of the condition treatment should be prophylactic Every patient about to receive horse serum should be questioned about susceptibility to other allergens and regarding previous serum treatment The patient should be tested with an intracutaneous injection of 0 01 cc of 1 100 dilution of horse serum If a wheal appears desensitization may be carried out by injecting 0 025 cc of 1 100 horse serum subcutaneously and doubling the dose every 30 minutes until 1 cc has been given If no severe reactions occur 0 1 cc of the serum is given in 5 cc of saline intravenously The do e is doubled every 20 minutes until the full dose is administered

Whenever serum is to be injected a syringe containing 1 cc of 1 1 000 epinephrine should be at hand and the full dose given intramuscularly on first indication of a reaction and repeated as required

ALLERGIC DERMATOSIS AND PHYSICAL ALLERGIES

Antihistaminic drugs orally will often allay the itching and swelling associated with these conditions Topical use should be avoided for sensitization to the antihistaminics themselves can develop

DRUG AND FOOD ALLERGY

Sensitivity to drugs and foods may be manifest by anaphylactic shock asthma angioedema or dermatoses The treatment for these reactions is the same as that listed on preceding pages

INSECT SPIDER AND SNAKE BITES

Bee Sting

Severe stinging by bees and wasps may constitute an emergency especially in hypersensitive individuals When multiple stings have been sustained severe swelling of the individual area occurs with intense pain

TREATMENT Usually the application of magnesium sulfate compresses is all the treatment that is necessary However highly sensitive individuals *may go into shock The hypotension is combated by* giving ephedrine 15 60 mg (¼ 1 gr) orally or 10 30 mg (⅙-½ gr) intramuscularly Epinephrine 0 5 cc subcutaneously also may be used as in the treatment of asthma (p 208) and repeated as necessary

SPIDER BITE

The black widow spider has a venom sufficiently potent to endanger human beings The venom causes dizziness weakness shallow respiration and increased blood pressure abdominal pain and rigidity

TREATMENT The species specific antivenin Lactrodectus mactans® is given in a 2.5 cc dose intramuscularly Before this horse serum antivenin is used the patient should be tested for sensitivity (p 214) When the antivenin is not available the acute symptoms require immediate treatment The administration of 15 mg (¼ gr) or more of morphine may be necessary to control the pain The spasticity may be controlled by intramuscular injection of 20 cc of 10 per cent solution of magnesium sulfate

SNAKE BITE

There are only a few poisonous snakes in North America namely the rattlesnake water moccasin copperhead and coral snake The symptoms vary with the toxin injected

TREATMENT When an individual is bitten by a snake a tourniquet is applied above the bite The wound should be incised and forced suction applied to promote free flow of serum The tourniquet must be released at intervals to prevent gangrene of the parts Antivenin should be obtained and injected as soon as possible One half of the contents of the vial

is injected at the site of the bite and the rest intramuscularly Repeat the injections at 1 to 2 hour intervals unless symptoms are strikingly decreased If the condition is extremely serious at the time of administration of antivenin it may be given intravenously A polyvalent antivenin marketed in 15 cc vials is available Every doctor practicing in regions where poisonous snakes occur should know of the closest place where antivenin is available General supportive treatment including saline intravenously blood transfusions and oxygen administration may be indicated

8

Emergencies Due to Central Nervous System Stimulation

CONTROL OF THE SEVERELY AGITATED PATIENT

THE PHYSICIAN is often called on to sedate a patient who is in a state of excitement which is otherwise uncontrollable These patients include persons with an acute and active psychosis or in delirium and alcoholics with delirium tremens

GENERAL CONSIDERATIONS

The patient is found by the physician *either in* complete and unchallenged possession of a wrecked household or *in involuntary restraint* by *friends* relatives or policemen The diagnosis of the excitement state is *seldom difficult and depends on the* available history Diagnosis of the cause of the excitement is not *essential* before the acute *manifestations* are controlled

Capture of the patient if he is still unrestrained is essential to control of the excitement Short of maneuvering of the patient into a convenient gas chamber, there is no long range means of subduing these excitement states It is pertinent to point out that there is no particular valor in being brave in these circumstances and the physician should insist on plenty of help in subduing the patient When the patient finally becomes accessible to medication the optimal approach is the administration of short acting depressant drugs intravenously By intravenous administration the dose can be adjusted relatively accurately and sedation is obtained promptly After exhausting efforts to reach an excited patient an erroneous idea often develops regarding the amount of drug necessary to subdue him and if the medication is given subcutaneously or intramuscularly overdosage is apt to result and distress again prevails over this new undesirable condition of the patient

Hypoxia —The patient may have been precipitated into an agitated or excited state by hypoxia and the alert diagnostician is careful to avoid using depressant drugs to cover the excitement and disorientation caused by lack of sufficient oxygen Close checking is imperative to determine if hypoxia from any source might have been the cause of the excitement or at least a perpetuating factor and after the excitement is controlled by a drug oxygen in excess should be supplied to correct any suspected deficiency In many agitated patients particularly in the postoperative and postanesthetic period the administration of excess

oxygen alone controls the excitement Depressant drugs are not necessary and if given tend to potentiate the hypoxia

Pain —Some excited patients (and again those in *the immediate postoperative period are subject to this* type) are agitated because of pain in the presence of residual anesthetics or hypnotics It is wise in these patients to determine if possible whether pain is an inciting factor and in this instance give an analgesic instead of a hypnotic to control the excitement In all other instances i e when there is no pain analgesics such as morphine are contraindicated for control of excited patients Hypnotics do a better job and for equivalent degrees of sedation produce much less respiratory depression

Available Drugs

BARBITURATES These are the most convenient drugs for sedation by intravenous administration The most commonly used are thiopental (Pentothal®), *pentobarbital sodium* (Nembutal®) *amobarbital* (Amytal®) secobarbital sodium (Seconal®) and phenobarbital Thiopental and pentobarbital sodium are to be preferred particularly when diagnosis is important because their action is relatively short and the patient is accessible for history taking and physical examination in a reasonably short time If the diagnosis is known and those in contact with the patient know that the excitement state may be protracted it may be better

to use the longer acting barbiturates such as amobarbital and phenobarbital

THIOPENTAL The dose needed for sedation depends on the patient's age and physical state and the degree of the excitment The last factor is often misleading and relatively small amounts of a drug may be needed to effect sedation in apparently very active states In the average middle aged adult in good physical condition an initial dose of 150 mg (6 cc of a 2½ per cent solution) of thiopental will induce sedation If this is insufficient the drug should be added in increments of no more than 50 75 mg (2 3 cc) until hypnosis is achieved It is wise to wait between additions of the drug for 1 minute by the clock to allow time for development of its effects and to avoid overdose One minute by the clock is quite a long time in the circumstances but the urgency of the situation seldom requires faster administration and the hazard of overdose is appreciably reduced Thiopental is relatively stable when mixed and ready for injection but it is not conveniently carried in the doctor's bag in the prepared state and time is consumed in mixing the powder with distilled water for injection

PENTOBARBITAL SODIUM SECOBARBITAL SODIUM More convenient barbiturates for injection in these situations are pentobarbital sodium or secobarbital sodium When administered intravenously they are quick acting and although the period of sedation is prolonged over that of thiopental it is not excessive rarely exceeding 2 hours Pentobarbital sodium

and secobarbital sodium for intravenous administration are prepared in ampules or rubber stoppered 50 cc vials in a concentration of 50 mg per cc It is quite stable in this preparation and can be carried in the doctor s bag for weeks without deterioration

The initial dose of pentobarbital sodium or seco barbital sodium in the average middle aged patient in good physical condition is 120 180 mg (2 3 gr) When administered intravenously they begin to act within 3 minutes and it is advisable to wait this length of time before administering additional amounts If more is needed to effect sedation the drug may be added in increments of 60 mg (1 gr) with ample time between additions

The dose of thiopental pentobarbital sodium or secobarbital sodium is decreased if the patient is a child or an elderly or debilitated individual There is little occasion for increasing the initial dose over that recommended but one can expect in the severely ex cited and the robust patient that several additions to the initial dose will be necessary

AMOBARBITAL (AMYTAL®) This can be administered in a 10 per cent solution The initial dose for the average middle aged adult in good condition is 180 300 mg (3 5 gr) It has the same disadvantage as thiopental in emergency situations—mixing of the powder and the diluent is necessary immediately be fore use The drug can be administered in increments of 120 mg (2 gr) and full effect can be expected within 3 to 5 minutes It is rather common practice to give

an initial dose of 450 mg (7½ gr) intravenously because the standard package of the drug contains this amount It must be emphasized that it is poor practice generally to give a single relatively large dose initially and to have that dose dependent on the amount of drug in the standard package Administration of drugs in this manner will many times achieve the desired depression but often will also result in excessive and alarming depression which is detrimental to the patient s welfare When any depressant drug is administered intravenously it is wise to take full advantage of the method and give the drug in relatively small initial doses to which are added only needed small increments up to the development of the desired state

PHENOBARBITAL SODIUM Phenobarbital sodium (Luminal®) can be administered intravenously in a 10 per cent solution A satisfactory initial dose is 120 mg (2 gr) To this can be added 60 mg (1 gr) increments as needed About 10 minutes is required to approximate the total effect of the drug and administration of succeeding doses at shorter intervals may result in overdosage

Depression caused by amobarbital and phenobarbital sodium persists in the average patient for 6 12 hours and the drugs therefore are convenient agents if protracted depression is desired

APOMORPHINE A drug infrequently used but a useful one for sedation is apomorphine It has good depressant qualities in addition to its ability to induce emesis and is highly satisfactory for intravenous ad

ministration in an emergency for prompt control of agitation It is particularly valuable in the treatment of the belligerent alcoholic or the patient with delirium tremens It may be given in doses of 1 2 mg (1/50 gr) or more and repeated within 15 minutes if necessary Overdosage results in significant respiratory depres sion which as always is controlled by active ventila tion of the lungs with oxygen The physician should not forget this useful agent for control of excited patients

The new drugs categorized as tranquilizers may be used for sedation especially in elderly patients This group includes chlorpromazine (Thorazine®) meprobamate (Miltown® Equanil®) and promethazine (Phenergan®)

Chlorpromazine may be given orally or intraven ously 0 100 Gm is a reasonable initial dose per os 0 025 Gm is a satisfactory dose intravenously One must be alert to hypotension especially if the drug is given intravenously Since this drug potentiates the ac tion of other depressant drugs, it must be given cau tiously if other drugs have been given The oral dose may be significantly increased in more agitated patients Long administration has been associated with biliary stasis and jaundice

Meprobamate is given per os A satisfactory dose is 0 200 Gm 2 to 3 times daily In the more severely agitated patients larger doses may be tried

Promethazine may be given intravenously in 0 025 Gm doses It should be remembered that this drug augments the depressant action of other drugs

PARALDEHYDE Two of the more common drugs used especially for the sedation of the moderately and more or less chronically excited or disoriented patient are paraldehyde and chloral hydrate Both drugs are excellent hypnotics and produce significant degrees of depression for protracted periods

Paraldehyde can be administered by mouth with fruit juices or by rectum alone or in combination with liquid petrolatum Despite its rather characteristic dis agreeable odor and taste it is apparently tolerated fairly well by many patients particularly chronic al coholics when taken by mouth It is given in doses sufficient to produce sleep from which the patients can be aroused by stimulation Its effect usually lasts 4 to 6 hours and is not accompanied by significant respiratory or circulatory depression provided the air way is unobstructed Oral or rectal dose for adults is 5 cc per 8 kg (1 drachm per 14 lb) This is consid erably in excess of the U S P dose but is necessary to establish the desired sedation particularly in the excited patient Paraldehyde may cause small widely dis seminated hemorrhages in the lungs which disadvan tage is a potent deterrent to its use in elderly and debilitated patients who have a predisposition to pul monary complications The drug may be given in travenously in doses of 1 2 cc at a time but one is reluctant to use the drug in this manner when one observes the black tarry sticky material that results from aspiration of blood into the syringe filled with paraldehyde It is probable that except in the few

patients who are not responsive to other depressant drugs or become more excited on administration of the barbiturates, paraldehyde will be used less and less for control of the excited patient

CHLORAL HYDRATE This is administered by mouth or by rectum It is somewhat toxic to the liver and continued administration for a number of days causes appreciable liver necrosis particularly when in sufficient attention is paid to the sufficiency of the oxygenation of the patient It is used to produce light stages of sleep and can be given in doses of 1 Gm (15 gr) and repeated every 4 to 6 hours if necessary It does not cause much respiratory depression if the airway is maintained and hypoxia does not supervene It is not too useful nor is paraldehyde for controlling the patient in extreme agitation of an emergency nature and its use will be limited to the perpetuation of sedation In these instances one can anticipate liver damage and may prefer to substitute some other depressant drug

BROMIDES In the mildly agitated patient sufficient and protracted control can be effected by the administration of bromides These are given in doses of 1 Gm (15 gr) orally and may be repeated However, it is well to remember that there is significant accumulation of bromides and when sedation must be maintained for days it is wise to reduce the dose or discontinue the drug altogether and substitute another After protracted administration of bromides a psychosis or dermatitis may develop During administration

of the drug it is imperative that the fluid intake be kept at a high level

TRIBROMOETHANOL WITH AMYLENE HYDRATE (AVERTIN®) This is a long acting drug which is almost always introduced per rectum It is useful when protracted sedation is required Narcosis of 4 to 6 hours duration can be anticipated The drug is given in a 3 per cent solution in distilled water and the dose of 100 mg per kg may be calculated in the following manner The patient s weight in pounds is multiplied by the factor 1 5 to give the total volume of 3 per cent solution Three per cent of this figure will give the number of cubic centimeters of the Avertin® fluid to be added to the total volume The distilled water is heated to 40°C (104° F) heating above this point will cause decomposition A few minutes of shaking will result in complete solution of the fluid in the water After mixing the solution is tested with a few drops of Congo red dye which is supplied with the package If the dye remains red the solution is all right for use if the dye turns blue or violet the drug has decomposed and the solution should be discarded

The solution is administered into the rectum a distance of approximately 10 cm (4 in) and care must be taken to see that the solution is retained The drug begins to take effect in about 10 minutes and the full effect is developed in about 30 minutes As in all states of significant drug depression care should be taken of the airway and the circulation

Large doses may precipitate hypotension If this is severe vasopressor drugs may be needed

The dose may be reduced if lighter degrees of sedation are desired In this situation the 100 mg per kg dose may be computed and appropriate per centages of the total volume selected Three per cent of this reduced volume can then be calculated

Children and particularly those in agitated states or in convulsions tolerate comparatively large doses although relatively small doses are all that are neces sary to control the spasms of tetanus

The maximal single dose is usually 100 mg per kg of body weight

Hydrotherapy and Ice Packs

Another effective means of controlling the agitated patient is the use of the ice pack or the cold hydro therapy tub The tub is likely to be available only in a large hospital whereas the ice pack can be used in the home

The patient is wrapped in sheets wetted with cold water When placing the sheets around the patient be careful to avoid constricting the chest Then place ice around the patient over the sheets In many in stances it is not necessary to use ice but merely to place the cold wet sheets around the patient The sheets should be changed at regular intervals, if left in place for long periods they become warm and since they are placed so closely around the patient

may induce hyperthermia from interference with radiation of heat from the body

CONCLUSION

The physician called on to control the excited patient will achieve most prompt and satisfactory results in the emergency if he administers the depressant drug intravenously In this regard he must remember that it is wise to give the drug in small increments until the desired depression is obtained The demand on the physician in these conditions is such that there is always a temptation to give too large an initial dose of depressant drug and this leads to subsequent difficulty in overcoming the unwanted excess depression The physician must always be alert to the airway and take measures to control it and provide oxygen if the sedation is of such magnitude that narcosis and relaxation of the jaw take place

CONVULSIVE DISORDERS

Convulsions are almost invariably emergencies and usually require prompt and efficient management Often this involves the use of relatively large doses of drugs and the physician must balance the danger of the drug against the severity of the convulsions There are a great many causes for convulsions and unless the physician is immediately aware of the direct cause of the convulsive seizure the indicated

procedure is to treat the condition as a symptom Symptomatic therapy involves use of the sedative drugs as discussed earlier in this chapter If the direct cause is known, some special types of therapy may be applicable For example for hypoglycemia and ionized blood calcium deficiencies specific therapy is indicated When faced with convulsions induced by certain poisons it is important to remember that many of the poisons exert a strong depressant action after the initial stimulation Therefore treatment must be directed to control only those of the stimulatory phase for long acting depressants of central nervous system may be additive with the depressant action of the poison (see Chapter 4)

In the control of convulsions it is seldom necessary to push the use of sedative drugs to the point of unconsciousness and just sufficient dosage is preferable to a large single dose In addition to controlling the convulsions the physician must protect the patient from bodily injuries and the tongue from injury due to biting A wedge made of rubber or tongue blades wrapped with adhesive tape prevents the tongue injury One must be careful that the tongue does not obstruct the airway (p 19)

Convulsions in Children

Convulsive seizures occur frequently in infants and *young children particularly with the onset of an acute* infection or a metabolic disturbance Convulsions caused by severe infections intracranial hemorrhage

convulsant drugs and many of the metabolic disturbances such as hypocalcemic tetany alkalosis and hypoglycemia are usually acute and self limiting Other types of convulsions such as idiopathic epilepsy are likely to be chronic and recurrent In newborn infants intracranial birth injuries and congenital defects of the brain are the usual cause of convulsions Acute infections and tetany are less likely to be the cause of the seizures in very young infants In early childhood acute infections are the most important causes of convulsions Beginning about the third year of life and continuing into later childhood idiopathic epilepsy is the commonest cause of convulsive seizures Less frequent causes of convulsions in infants and children are tetany hypoglycemia poisoning asphyxia nephritis with uremia and degenerative diseases of the central nervous system Every effort should be made to determine the exact cause of the convulsive seizure so that ultimately proper therapy may be aimed at the underlying cause The age group and history usually give some insight into the cause Figure 34 shows the comparative incidences of various types of convulsions at different ages

TREATMENT When the doctor is called on the telephone to attend a child in a convulsion, the mother should be advised to give the child or infant a warm tub bath Often this simple procedure causes a mild convulsive attack to subside and at least it keeps the anxious parents busy until the doctor arrives If an acute infection is presumed to be the

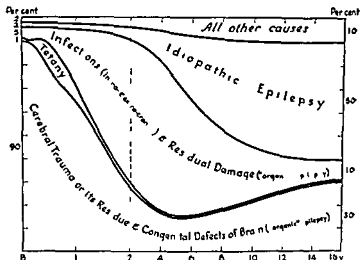


FIG 34 —Comparison of causes of convulsions from birth to 16 years based on data from two general children s clinics in the northern part of the United States between 1925 and 1940 The percentage distribution is not universally applicable nor does the chart represent incidences of different causes for a much earlier period in the same areas because preventive measures have greatly reduced the frequency of infantile tetany and acute infectious diseases in the past two decades (From Nelson)

cause of the convulsive seizure sedative and antipyretic measures may be used If the convulsion is severe thiopental or a short acting barbiturate (as previously described) should be given In less strenuous circumstances and to diminish the possibility of recurrence a hypodermic dose of 15 30 mg (¼ ½ gr) of phenobarbital may be given

Prompt administration of an antipyretic agent such as acetylsalicylic acid acetanilid or acetophenetidin may prevent the convulsions often associated with high fevers Smaller doses of phenobarbital usually suffice in the milder types of convulsive disorders However severe convulsions such as may occur in measles and meningitis may last several hours and require heavier sedative treatment Tribromoethanol 80 mg per kg of body weight may be given rectally or phenobarbital sodium by vein in dosages varying from 60 mg (1 gr) for infants aged 6 months to 1 year to 120 mg (2 gr) for older children Intravenous barbiturates must be administered slowly as rapid injection causes a precipitous fall in blood pressure Adequate oxygen therapy or other means of resuscitation should be readily available In prolonged convulsions hypoxia may be a complicating factor and oxygen should be administered After the convulsive seizures have been abated information regarding the etiology of the convulsions should be secured so that appropriate subsequent therapy may be instituted

Convulsions in Newborn

Convulsions in the newborn are most often associated with brain damage due to anoxia or trauma If the convulsion is due to one of these conditions administer 15 30 mg (¼ ½ gr) of phenobarbital sodium hypodermically If the convulsion is due to hemorrhagic disease of the newborn a blood transfusion is indicated to stop the bleeding For convul

sions that may be caused by hypoxia associated with congenital heart disease or atelectasis adequate oxygen therapy is indicated

CONVULSIONS IN ADULTS

Convulsions occur less frequently in adults than in children but the emergency is of equal importance Protection from physical injury and use of sedative drugs are the emergency measures to be employed When first seen the patient should be given an intravenous injection of 60 180 mg (1 3 gr) of pentobarbital sodium or 150 500 mg (6 20 cc) of a 2½ per cent solution of thiopental An airway should be available to combat any respiratory difficulty The longer acting phenobarbital sodium 150 300 mg (2½ 5 gr) may be given very slowly intravenously Its action is not as prompt as that of the more rapid acting thiopental The use of morphine as a sedative is contraindicated in most convulsions owing to its respiratory effects For convulsions due to poisons such as strychnine atropine and cocaine a relatively short acting barbiturate such as thiopental or pentobarbital sodium should be used so that severe depression does not outlast the stimulating phase

Epilepsy —In the isolated seizure of epilepsy practically all the physician can do is to protect the patient from bodily injury and protect the tongue by a wedge of wood or cork placed in the mouth Additional treatment is that used in the general management of epilepsy The convulsive phase is self limiting and needs

no specific therapy Status epilepticus may require the use of tribromoethanol in doses of 100 mg per kg Long acting barbiturates such as phenobarbital sodium also may be useful, it may be administered intra venously Rectal administration of chloral hydrate or bromides may control the convulsions if given at about two hour intervals Dosage depends on the patient s age For convulsions known to be associated with allergies the best drug to use during the individual seizure is 1 1 000 epinephrine 0 5 cc intramuscularly

Tetanus—For the convulsions that occur in tet anus tribromoethanol 80 100 mg per kg of body weight given rectally is the most satisfactory agent Smaller initial doses are often effective, they may be repeated as needed to control the tetanic spasm

Hypoglycemia—For hypoglycemic convulsions glucose or other carbohydrates should be adminis tered at regular intervals

Acute poisoning—Convulsions due to poisons are treated according to the specific causative agent (see Chapter 4) Until the poison is identified the general procedure is to treat the symptoms as they arise fol lowing the general procedure outlined for control of convulsions (p 229)

Eclampsia—Morphine is apparently the drug of choice for eclamptic convulsions The patient who has had or is about to have a seizure is given 15 mg (1/4 gr) of morphine sulfate One hour later she may be given 2 Gm of chloral hydrate by rectum if un conscious or by mouth if conscious The rectal ad ministration is made in 100 cc of physiologic saline

and the oral administration in milk The morphine is repeated in about 3 hours and the chloral hydrate in 7 hours Although the induction of hypotension (relative) by such means as spinal or epidural analgesia and hexamethonium may seem heroic, there may be merit in this more vigorous method of therapy The level of spinal or epidural analgesia must be in the neighborhood of T6 in order to paralyze the splanchnic nerves and promote hypotension If the hypotension becomes excessive it may be corrected with vasoconstrictors, given intravenously in small repeated doses Hexamethonium may be given intravenously in doses of 10 mg at 5 minute intervals until a satisfactory reduction in arterial tension is effected It may be repeated as needed to maintain the lowered tension If the hypotension exceeds the desired level, it may be corrected with vasoconstrictors

It is particularly important that the airway be watched closely and oxygen therapy and suitable ventilation be administered if the depression is severe and prolonged

Delirium tremens—This type of acute insanity occurs fairly frequently in heavy drinkers and the diagnosis is relatively easy The best sedative drug for its treatment is paraldehyde An initial dose of 30 cc (1 oz) is given orally and followed by 15 cc (½ oz) every hour until controlled It may also be given rectally, mixed with equal parts of liquid petrolatum as a retention enema Thiopental in small doses intravenously offers good control but it is difficult to make the injections in these excited patients Morphine

should not be given because it tends to produce respiratory insufficiency Additional treatment consists of sodium chloride administration to combat the dehydration a high calorie, high vitamin diet and plenty of fluids Other complicating symptoms must be treated as they arise

Tetany—In tetanies associated with low ionized calcium the treatment is directed toward increase of the calcium content of the blood If actual convulsions occur they may be controlled temporarily by sedative drugs and later by oral administration of calcium chloride The initial dose of calcium chloride is 2-5 Gm dissolved in water followed by 1-2 Gm every 5 to 6 hours Its acidity promotes absorption and the tendency toward acidosis aids in the calcium ionization in the blood Equally good results are obtained with calcium lactate in water if given in about 1½ times the dose of the chloride If oral administration is not feasible calcium gluconate 10 per cent in doses of 2-10 cc may be given intramuscularly Occasionally this agent leaves persistent nodules at the site of injection In severe convulsions with spasm of the glottis 2 5 cc of 10 per cent calcium gluconate may be given intravenously The injection must be given very slowly to avoid nausea and vomiting Rapid administration may cause acute paralysis of respiration and circulation

9

Care of Patient in Acute and Chronic Comatose States

ACUTE COMATOSE STATES

Acute Drug Depression

THE PATIENT with a drug depression poses an acute problem which can often be solved provided certain measures are taken immediately and a definite chronologic order is established and followed If the resuscitative measures are haphazard and important steps are overlooked or sidetracked many patients will be lost unnecessarily

PROCEDURE The following paragraphs outline briefly the treatment for the patient with an acute and severe drug depression

Step 1—As soon as the patient is seen the first procedure is the securing and establishment of a patent airway During this step, the character of the respiration can be determined Note should be made of the rate depth and presence or absence of accessory

respiratory muscle activity and whether or not the intercostal muscles and diaphragm are both functioning These observations will be of assistance later in the diagnosis of the type of depression

Step 2—Following establishment of the airway the extent of ventilation is determined If pulmonary ventilation is inadequate the patient must be assisted by the most suitable means at hand Carbon dioxide should not be given in any concentration Carbon dioxide therapy is not needed by the patient suffering from drug depression, because depression of respiration results in a retention of carbon dioxide in the blood stream sufficient for stimulation of the well oxygenated respiratory center Carbon dioxide given in an attempt to stimulate even the well oxygenated but drug-depressed respiratory center frequently causes even greater depression and may provoke convulsions Carbon dioxide will on occasions stimulate respiration in the depressed patient but the stimulation is brief the margin of safety is minimal and the use of this agent is physiologically questionable

In the light of evidence on the deleterious cardiac effects of abrupt reduction in elevated carbon dioxide tensions one wonders about the possible disadvantages of artificial ventilation with only oxygen atmospheres However it seems that it might be more expedient to adjust ventilation and associated elimination of carbon dioxide in a patient requiring artificial ventilation than to make guesses as to the optimal amount of carbon dioxide to add to respired atmospheres under these circumstances

Step 3 —After the patient s airway has been established and ventilation improved the next step in treatment is directed toward determining the state of the circulation and correcting its deficiencies Most patients with a major drug depression suffer from varying degrees of shock, either neurogenic or secondary, depending on the degree of depression the drug causing it, the degree of hypoxia and the length of depression before treatment Treatment should be promptly and thoroughly instituted according to the principles outlined in Chapter 3

Step 4 —Until now with the exception of the observations made regarding the respiration no direct attempt has been made to establish the diagnosis and to institute specific therapy Specific therapy has not been indicated even though the nature of the drug causing the depression is known Patients will die for lack of the prompt institution of the foregoing measures particularly measures concerned with respiration and circulation but they will not die for lack of early specific therapy

Steps can now be taken to determine the cause of the depression A thorough physical examination should be made The examination of the eyes is important Pinpoint pupils are a characteristic of morphine and other narcotic depression Pupils in mid dilatation are most frequently associated with barbiturate poisoning Unequal pupils are often indicative of intracranial lesions During care of the airway, the breath will have been noted and the presence or absence of diabetic acidosis and alcohol determined Thorough

search should be made of course, for fractures bruises needle marks etc

Assuming that the depression is due either to a narcotic or to one or more of the barbiturates the examiner now has several diagnostic aids With the additional aid of a history it is usually possible to establish a provisional diagnosis which later may be confirmed by laboratory investigation of stomach and bladder contents

Step 5 —After the airway has been taken care of the patient ventilated the efficiency of the circulation restored and more specific diagnostic and thera peutic efforts made the stomach should be emptied with a large stomach tube Small bore stomach tubes such as the Wangensteen tube are inadequate for this purpose because they fail to remove the larger food particles Extreme care should be exercised to prevent aspiration of stomach contents during this procedure It is imperative that suction apparatus be immediately at hand The stomach contents should be saved for examination in the laboratory

Step 6 —After aspiration of the stomach the blad der should be catheterized and the urine saved for examination for the presence of the offending drug

Step 7 —If the depression is due to a narcotic n allylnormorphine hydrochloride and levallorphan tartrate may be used as outlined (see Chapter 1 page 34) If the depression is due to a short acting barbit urate the patient requires only good nursing care and perhaps fluid and nutritional additions The effective

ness of Megamide® is still debatable If effective its action may be no more than that of a non specific analeptic such as pentylenetetrazol or nikethamide If however, the barbiturate is one of the long acting group, there is a slight advantage in maintaining basic reflex activity by the use of picrotoxin Picrotoxin is given intravenously at the rate of 1 mg per minute until the patient regains pharyngeal and laryngeal reflexes and muscle tone or until muscular twitchings appear The latter usually appear first around the face but they may show up earlier in the extremities An attempt should be made to maintain a reasonable degree of reflex activity and tone by repeated intra muscular injections of picrotoxin No attempt should be made to bring the patient to full consciousness al though he may respond to painful stimuli If by mis judgment the dose of picrotoxin is too large and con vulsions ensue an intravenous injection of a barbiturate must be given immediately to control the convulsions When the dose of a long acting barbiturate has been large the depression may persist for several days Good nursing care is essential to minimize pulmonary complications Caloric intake may be accomplished by parenteral means

Diabetic Coma

The prompt recognition of diabetic coma is ex traordinarily important because specific therapy is available *Often* the diagnosis is obvious because the patient is known to be a diabetic and available in

formation indicates that he has broken his diet or failed to take his insulin or that he is suffering from an infection The patient in diabetic coma is dehy drated the eyeballs are soft respiration is of the Kussmaul type the odor of acetone is often apparent on the breath vomiting frequently occurs the pulse is rapid and blood pressure low abdominal findings may suggest appendicitis and the pupils are dilated The presence of several of these findings in a coma tose patient should immediately suggest diabetic coma

Steps 1 2 and even 3 outlined in the preceding section are occasionally necessary and can be followed while confirmatory diagnostic tests are awaited The patient should be hospitalized whenever possible so that determinations of blood sugar and plasma carbon dioxide and urinalysis can be done He should be kept warm and as soon as the diagnosis is made re ceive 20 100 units of regular or crystalline type insulin even before transfer to a hospital and the determina tion of the essential laboratory data All of the insulin is ordinarily given subcutaneously Additional doses of insulin and frequency of administration depend on the severity of the acidosis The details of treating this coma after the initial emergency are beyond the scope of this discussion As a part of the emergency therapy arrangements should be made to hydrate the patient and the stomach should be emptied and lavaged through a stomach tube Frequent urinalysis so im portant in caring for such patients is facilitated by installation of an indwelling catheter in the bladder

Hypoglycemia and/or Insulin Shock

This acute coma responds rapidly to parenteral restoration of an effective blood sugar level and admittedly it is imperative that this therapy be given as early as possible Nevertheless, it is re-emphasized that care of the airway supply of oxygen and an evaluation of the circulatory status is still essential, especially if the diagnosis is not immediately apparent The symptoms of hypoglycemia are the same whether the condition is induced by insulin overdosage or occurs spontaneously The real hazard is that the unconsciousness of hypoglycemia be confused with that of diabetic coma and insulin be given For this reason determination of the blood sugar is advisable before specific therapy is instituted The patient who is unconscious because of hypoglycemia is likely to be sweating and he may have convulsions This acute coma responds dramatically to intravenous administration of glucose The response is so prompt that often before 10 cc of a 50 per cent solution has been administered recovery has occurred Once the emergency is past definitive therapy must be undertaken This may be quite simple if the condition is due to insulin overdosage in the diabetic but if it is of the spontaneous type an etiologic diagnosis must be made This can be a difficult problem details of which are beyond the scope of this volume

Uremia

The patient with advanced uremia may be in more immediate serious difficulty because of lack of a patent

airway sufficient oxygen and competent circulation than he is from the disorder precipitating the comatose state When indicated attention should be given these points as outlined at the beginning of this chapter The treatment of uremia is usually disappointing because of the advanced renal failure which precipitates it When it is due to urinary retention as in benign prostatic hypertrophy continuous catheter drainage and attention to fluid and electrolyte balance usually bring striking improvement Occasionally when renal disease is the precipitating factor correction of such extrarenal factors as dehydration and disturbances of acid base balance produces temporary improvement these details are not emergency measures Convulsions of uremia are at times controlled by reduction of elevated cerebrospinal fluid pressure or by administration of magnesium sulfate 2 0 3 0 Gm (30 45 gr) intramuscularly or 20 25 cc of a 10 per cent solution intravenously

PROTRACTED COMA

The general practitioner is often responsible for the welfare of a patient who remains comatose from a cerebral vascular accident uremia trauma accidental or suicidal overdose of depressant drugs or other causes for long periods It is a difficult situation in the hospital and even more difficult in the home However a few fundamental principles apply in either the home or the hospital that make the care

of the chronically comatose patient less of a problem and safer for the patient The care of the airway oxygenation of the patient and attention to the circulatory status remain important features in chronic comatose states

Bedsores—Prevention and Treatment

In all such patients particularly elderly ones there is the constant menace of the decubitus ulcer or bedsore These unsightly and dangerous lesions are nearly always preventable by adequate nursing care Measures for control of these lesions and their prevention must be taken promptly because they may develop within 12 hours and once established are most refractory Because the ulcers are due largely to ischemia it is essential that no area of the body and particularly prominent bony eminences be exposed to pressure for any appreciable period The patient must be turned frequently and whether in a household or in a hospital this calls for a rigid time schedule For convenience a schedule that outlines turning of the patient from back to right side from right side to left side from left side to prone and from prone to back every 2 hours night and day will aid the prevention of the ulcers The family and the nursing assistants must be thoroughly impressed with the necessity for maintenance of this rigid schedule This turning procedure minimizes the possibilities for development not only of ulcers but also of pulmonary congestion and pneumonia The patient s skin must

be kept dry and well powdered Extreme care must be exercised in the turning maneuvers that no slight skin burns result from the rubbing of the patient s skin on bed sheets other coverings or clothing The sheets must be kept free from wrinkles Small and apparently insignificant abrasions may be the beginning of large slowly healing ulcers It is wise also to be very careful that rubber sheets although necessary are not allowed to come in contact with the patient The patient s feet should not rest against the foot boards or post of the bed and they should not be maintained in extension by neatly tucked in bed covering

For the patient who is incontinent the area must be cleansed and dried after each soiling and small disposable pads kept in the area to prevent soaking of the bed clothes and mattress When the patient is likely to be comatose and incontinent for a long period it is wise to devise a sling arrangement which will keep the buttocks suspended and free from the soiling material Again if such an arrangement is used the area of contact with the sling must be especially well observed for evidences of early ischemia and necrosis The Gardner air cell mattress is so designed that no one area of the body is exposed to pressure longer than 5 minutes Air is alternately removed and then replaced by a pump Constant pressure is avoided and some beneficial massage accomplished If it is anticipated that the patient will remain comatose for a long time such a mattress would be desirable

CARE OF THE URINARY BLADDER

Adequate care of the urinary bladder is an extremely important part of the treatment of the patient in coma and should be instituted immediately otherwise the complications of inadequate care may ensue The three principal complications are (1) overdistention of the bladder with myogenic atonia and persistent residual urine with all its sequelae (2) infection of the urinary tract with resultant sepsis possible renal insufficiency and late occurrence of renal calculi (3) dribbling of urine from overflow or incontinence due to incompetence of the urinary sphincters resulting in excoriations of the genitalia thigh and buttocks and predisposing to rapid formation of decubitus ulcers To avoid these serious complications care of the urinary bladder of a comatose patient must be instituted as soon as possible This may be accomplished in several ways

CATHETERIZATION 1 If the sphincters are competent so that no incontinence occurs one may catheterize the urinary bladder regularly every 6 hours *If this can be done easily with careful aseptic technic* it is quite satisfactory It prevents overdistention of the bladder and allows for the rhythmic filling and emptying of the bladder There are two dangers of this method (*a*) Introduction of infection may occur with the repeated trauma to the urethra that is inevitable with this method (*b*) If catheterization is not done regularly and at intervals of not more than 6



FIG 35 — Patient with indwelling Foley catheter connected through a Y tube to a collecting jug and a flask containing antiseptic solution (1 : 0·000 $KMNO_4$) which can be used to irrigate the bladder at stated intervals Between irrigations the outflow tube from the flask is clamped off and the bladder kept draining continuously through the catheter to the jug

hours overdistention of the bladder may occur with consequent myogenic atonia after the primary disease has been overcome

2 One may immediately introduce an indwelling urethral catheter of the Foley type (Fig 35) This is highly satisfactory It keeps the bladder empty so that no overdistention occurs and avoids the urethral trauma caused by frequent catheterization so that infection is minimized It also carries the urine away continuously so that it may be used when the sphincter is incompetent The only disadvantage is that it does not allow for rhythmic filling and emptying of the bladder However this is of little importance if the need for this type of bladder care will exist for only a few days or one or two weeks, in this circumstance the Foley catheter is most satisfactory

3 If treatment will be necessary for more than two weeks tidal drainage with an indwelling urethral catheter has many advantages (Fig 36) In addition to avoiding trauma to the urethra and carrying the urine away so that no dribbling will occur it permits the rhythmic filling and emptying of the bladder, maintaining the tone of the bladder wall at its most normal condition However, it is more difficult to avoid infection of the urethra bladder and upper urinary tract with this method than with the ordinary indwelling urethral catheter

4 If severe infection of the urethra bladder or upper urinary tract supervenes *suprapubic cystotomy* must be done otherwise there will be irreparable



Fig 36.—Patient with indwelling Foley catheter attached to a tidal irrigator. The irrigating solution (1:10 000 $KMnO_4$) drips into the bladder at the rate of 15 drops a minute through the drip apparatus. As pressure in the bladder rises, the level of fluid in the ascending arm of the inverted U tube rises. When it reaches the desired amount, determined by the height above the bladder at which one places the U tube (a good place is 1½ ft), it crosses over into the descending arm, causing the bladder to empty by suction into the flask on the floor. When the bladder is empty, air is sucked in, breaking the suction, and the process is repeated. It is important that the diameter of the inflow drip apparatus be much smaller than that of the tubing used for the inverted U tube.

damage to the urinary tract This gives better drainage of the urinary tract through a nonabsorptive area and thus aids in combating infection

To keep down infection antibiotics and other chemotherapeutic agents should be used If these are not adequate when used with methods 1, 2 or 3, suprapubic cystotomy should be done Large doses of penicillin must be given if satisfactory results are to be obtained 1 200,000 units daily in divided doses is the usual routine Sometimes 5 Gm (75 gr) of sulfadiazine in normal saline is given twice daily in addition Streptomycin may be given at times in conjunction with penicillin in divided doses 1 Gm daily

Care of the Bowel

For the patient who is incontinent of feces and in a cast as well as being comatose, it is desirable to line the area around the buttocks with Koroseal It is relatively nonirritating and is easily cleaned The comatose patient s bowel movements must be checked carefully because of the tendency to fecal impaction A daily small dose of mineral oil is advisable and an enema should be given every 3 days if there are no movements in the interval

Fluid and Food Intake

The maintenance of proper and adequate fluid and food intake in the comatose patient is more easily accomplished in the hospital but can be done in the

home Often both fluid and food intake can be achieved by an indwelling Wangensteen catheter through which a liquid diet of the proper caloric and nutritional values can be placed on a regular schedule A recom mended diet follows

1st hour glucose water 120 cc (4 oz)
2d hour sweetened fruit juices 120 cc (4 oz)
3d hour eggnog 120 cc (4 oz)

The sequence is repeated throughout the 24 hours Three grams of table salt should be added to the daily diet along with adequate vitamin supplements

STOMACH CATHETER The placing of a Wan gensteen or Levin stomach catheter in the comatose patient is seldom too difficult even without co-opera tion by the patient It is wise to obtain a new cath eter or use one that is not flimsy and worn The catheter is lubricated with liquid petrolatum and in serted through the side of the nose that seems more free After it has been placed in the oropharynx it should be rotated so that the tip is directed toward the right side of the pharynx because the esophageal opening is usually on that side It is then moved for ward easily and persistently until gastric contents run out or can be aspirated from the end of the cath eter If resistance is encountered the catheter should be withdrawn and reinserted in a different direction If this is unsuccessful and the catheter continues to curl up in the pharynx it is sometimes necessary to use a laryngoscope (Fig 5 p 26) and place the catheter into the esophageal opening under direct vision Palpation of the catheter in the oropharynx

and its direction by the finger toward the esophageal opening is sometimes effective and can be tried before laryngoscopy The catheter may tend to enter the glottis and trachea instead of the esophagus This abnormal entry often can be detected if the patient coughs and air is heard passing through the catheter If this occurs the catheter should be reinserted after the tip of the catheter has been directed more to the right and posteriorly

There are several tests for the presence of the tip of the tube in the stomach other than the free flow of unmistakable gastric contents from the open end of the tube One can often detect its presence by placing a stethoscope over the stomach and listening for the borborygmi produced by introducing air from a syringe attached to the open end of the catheter One should be careful not to be misled by drainage or aspiration of brown mucus from the tracheo bronchial tree which may closely resemble gastric contents A common test is to place the open end of the catheter in a glass of water and watch for bubbles This is not reliable because the tip may be occluded with mucus that prevents free flow of air

If a Wangensteen or Levin catheter must remain in place for long periods, it is imperative that the patient be watched closely for evidences of ulceration in the nose naso and oropharynx and particularly the esophagus Low grade elevations of temperature not otherwise accounted for may be due to esophagitis Severe esophagitis can occur frequently

followed by perforation and subsequent mediastinitis

If it seems inadvisable to give fluid and food through a Wangensteen or Levin catheter in the stomach nutrition must be maintained by parenteral means either intravenously or subcutaneously Salt protein carbohydrate and vitamin intake can be maintained effectively by this route for long periods Technics of venipuncture and maintenance of long standing drip procedures are described in Chapter 11

Care of the Mouth and Eyes

The mouth and oral cavity must be supervised closely Crusts and herpetic lesions that develop on the lips should be removed with care and with all possible asepsis The lips and oral cavity should be cleaned regularly with half strength hydrogen peroxide or lemon and glycerin mixture 1 teaspoon (5 cc) of lemon to 2 tablespoons (30 cc) of glycerin and covered with glycerin The teeth should be scrubbed with either a brush or an applicator tipped with cotton If the fluid intake is maintained the incidence of parotitis will be low but this complication should be watched for It is less likely to develop when antibiotics are given early to minimize the possibility for infection here as elsewhere in the body

The eyes of the comatose patient are often wide or half open and should receive protection White s ointment can be applied and the eyes covered with moist sponges It is sometimes advisable to suture the eyelids closed

Prevention of Burns

Comatose patients are extremely susceptible to burns and great care should be taken to avoid burning the skin with hot water bottles or heating devices of any sort A temperature that is satisfactorily tolerated by other patients is often too high for the skin of the comatose patient This is particularly true when hot water bottles are placed over areas into which subcutaneous fluids have been introduced

Care of the Airway

The establishment of the airway and its maintenance were mentioned earlier but it is well to re emphasize the importance of constant attention to this detail Particularly in the chronically comatose patient suction is important for the removal of accumulated mucus from the pharynx and tracheobronchial tree This material if not removed seriously interferes with the patient s ability to maintain adequate oxygenation and predisposes to the development of atelectasis and pneumonic processes Therefore it is wise to establish a regular schedule for the removal of this material

CONCLUSION

With faithful adherence to the measures outlined for care of the comatose patient, many patients lives will be saved and many others will be in better physical condition for the period of the coma There is no

substitute for constant vigilance and supervision and there are no short cuts for the labor involved Proper care of the comatose patient is a difficult task but it is less of a task than the care of a patient who has been improperly supervised and is ridden with bed sores dehydrated and malnourished and rasping with pneumonia

10

Miscellaneous Emergencies

IN THIS chapter a number of miscellaneous but reasonably common and important emergency situations are discussed

GASTRIC DILATATION

Death from acute gastric dilatation particularly postoperatively is not uncommon but it is almost inexcusable The physician must be constantly aware of the possible development of this complication and be prepared to act at once because when recognized early and treated promptly and courageously it need not be fatal Gastric dilatation is often precipitated in the patient who is adept at swallowing air, and such a patient should be watched closely One must also be alert to this development in the comatose patient, because the gasping accentuated inspiratory efforts in some of the patients permit aspiration of air into the esophagus and down into the stomach

Early signs and symptoms of acute gastric dilatation are discomfort in the epigastrium lack of appetite

and a tendency to nausea Soon the patient begins to vomit small amounts of liquid material, but obtains no relief from the distress which includes an urgent desire to belch If dilatation is not relieved the patient continues to vomit and precipitously goes into severe and refractory circulatory collapse followed by death in a short time

Appearance of these signs and symptoms should prompt the physician to examine the abdomen and palpate percuss and auscultate for evidences of distention These include a large area of dullness topped by an area of hyper resonance from the gas a large palpable mass and a gurgling sound in the stomach area

TREATMENT Treatment is simple consisting in immediate placement of a stomach tube to which is attached a suction device for removal of the gas and fluid Recovery is usually prompt and complete If the stomach is very large it is advisable to remove the contents relatively slowly to avoid circulatory collapse The tube should be left in place to avoid recurrence of the dilatation The stomach takes some time to recover its normal tone and mobility after a bout of acute distention

If there is a question about the diagnosis nothing is lost by inserting a stomach tube and much may be gained

BURNS

In any case of severe burn all efforts should be directed at preventing death from shock which may

occur within the first 48 hours Unless the burn is due to chemicals, no attempt should be made to remove the burned garments or to clean the burned sites while the patient is waiting to be moved to a hospital The burned areas should be covered with clean sheets to prevent further contamination Demerol® or morphine is given to relieve pain

TREATMENT At the hospital the local burned areas are not treated until preventive measures against shock have been instituted This consists mainly in the intravenous injection of plasma A burn in an infant is more serious than in an adult because shock from a burn is related not to the depth of the burn but to the surface area involved The burned area on an infant naturally involves a greater percentage of the total body surface than on an adult and is therefore much more likely to result in shock Be wary of the patient who looks well he may suddenly go into vascular collapse A burned person who has an accompanying fracture or wound is more likely to go into shock than if there were no injury

Severely burned patients should receive tetanus antitoxin 1 500 units unless they have been previously immunized with the toxoid in which case a 1 cc stimulating dose of the toxoid is administered Penicillin therapy is started immediately preferably by the intramuscular route

For a minor burn one of the simplest of home remedies is well moistened baking soda Sterile dressings of petrolatum gauze are the most suitable The

use of proprietary preparations containing picric acid is condemned The patient may be allergic to picric acid and after application of picric acid ointment may have a much more severe burn than the one for which he was originally treated

Any burn involving the face may damage the eyes If this is suspected install a few drops of mineral oil or castor oil and have an ophthalmologist see the patient at once If the burn is due to alkali or acid wash the eyes repeatedly with tap water and have the patient examined by an ophthalmologist immedi ately Burns of the face may be accompanied by in volvement of the upper respiratory tree After a few hours the tissues may swell just as tissues on the body surface and cause respiratory difficulty This is es pecially true in children This complication may be treated with a cocaine spray (5 per cent solution) of the area The vasoconstrictive property of the cocaine causes a reduction in the edema and relieves the ob struction

SUNSTROKE

Sunstroke is an acute emergency which demands early diagnosis and active therapy to insure satisfac tory results

Sunstroke is characterized by the development of excessively high body temperature usually owing to failure of the sweating mechanism This results in an accumulation of heat in the body to the point where the heat regulating mechanism of the brain

stem becomes relatively inoperative and uncontrolled elevation of temperature occurs

The patient with a sunstroke has, in addition to the high temperature a hot dry skin and is frequently comatose and often delirious The pulse is extremely rapid and the patient is bright pink

TREATMENT The patient is immediately put in bed on wet iced sheets without night clothes and ultimately wrapped in ice Oxygen therapy must be instituted immediately to support the rapid heart and to insure adequate oxygenation of the brain stem to facilitate restoration of function in the deranged heat control area Fluids are not necessary and may be detrimental The ice pack is maintained until the body temperature declines to within about 3 degrees of normal then removed and the lowering of the temperature is accomplished more slowly If the packs are not removed until the body temperature is normal the temperature will decline more than is desirable or necessary Checking of the emperature every 5 minutes is essential

The patient may be delirious and the disorientation may interfere with the therapy If so small amounts of barbiturates may be given intravenously to control the agitation, but these agents should be used only if the packing does not control the delirium and should be given very cautiously Close observation of these patients is necessary for several hours after the temperature has been controlled because the heat regulating center may not return to complete function

for some time and there may be wide fluctuations of body temper iture that require external control

The use of drugs to precipitate sweating is inad visable and often entirely ineffectual

HEAT EXHAUSTION

Heat exhaustion is characterized by the develop ment of shock due to excessive fluid and salt loss from excessive sweating The condition can be recognized as that of shock and treated as such The patient except in the extreme phases is usually conscious He is pale perspiring profusely and has a weak thready and rapid pulse hypotension and relatively low body temperature

TREATMENT The patient is put to bed with a normal amount of covering for the environmental temperature Excessive covering and the use of hot water bottles or other heating devices accentuate the fluid loss and perpetuate the shock It is probably advisable to place the patient in a slightly head-down position Oxygen should be started immediately to help to prevent capillary ischemia and ultimate per-meability to plasma

Fluid for replacement is ideally normal saline since the fluid loss has been that of water and sodium chloride In the less acute situations water (not ice water) and coated salt tablets are often effective The water should be given in small often repeated amounts and the salt in 1 Gm (15 gr) tablets 2

tablets immediately and 1 tablet every half hour for 2 hours If it is not advisable or is impossible to correct the deficiency by oral medication intravenous administration of normal saline is necessary It can be run in rather rapidly for the first 15 minutes or until the patient stops perspiring whichever happens first and then administered slowly over several hours until 7-9 Gm (105 135 gr) of salt has been given More fluids will be required but the patient usually will have recovered sufficiently to augment the paren teral supply by drinking The patient should be watched closely during the recovery period for evidence of anuria or oliguria and fluid intake should be sufficient to produce a daily urinary output of 1 000 cc or more

As in sunstroke drugs are usually not indicated and the shock should not be treated with vasopressor drugs

11

Technics of Venipuncture

INNUMERABLE times in this text reference has been made to the advisability of and the necessity for the intravenous administration of drugs and fluids in emergency situations It is readily apparent that proficiency in venipuncture is essential to satisfactory solution of an emergency problem Every physician has been confronted with the frustrating and frantic experience of attempting and reattempting venipuncture when intravenous medication was imperative Such exasperating episodes will occur less frequently if certain simple but effective measures are taken to insure a higher percentage of successsful venipunctures

Figure 37 shows the superficial and deep veins most accessible to use for introduction of intravenous medication The physician will do well to memorize these sites and inspect them with care whenever the need for venipuncture arises The sites most often used are the antebrachial veins the veins on the dorsum of the hand and the veins just anterior to the medial malleolus of the tibia at the ankle

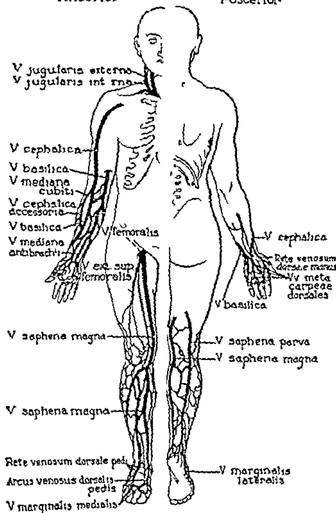


FIG 37—Deep and superficial veins accessible to venipuncture (From Lundy)

METHODS FOR DILATING VEINS

Many times when intravenous medication is indicated and sorely needed the veins are collapsed and well nigh invisible There are two methods by which these veins can be made visible and reasonably accessible to puncture

1 The first method consists of the use of heat although in emergencies there is often not enough time to achieve dilation by heat The method is used principally on the veins of the extremities Hot moist bath towels are placed directly on the skin then covered with rubber sheeting or other impermeable heat retaining material Over this can be placed hot water bottles It is important that the toweling be placed over a wide area On the arm it should be placed from the hand to the mid upper arm and on the leg from the foot to the mid leg The extremity should be dependent while the heat is applied The tourniquet is applied while the heat is still on the dependent extremity To obtain the best results the heat should be left on for about 20 minutes although shorter periods are often sufficient Application of cold antiseptics should of course be avoided

2 The second method consists of dilation of larger veins by entry into a small vein and injection or saline distal to a tourniquet On the extremities small veins are usually visible but larger veins needed for the introduction of large volumes in a short space of time are often not readily recognized especially in patients in shock In these circumstances it is con

venient and time saving to place a tourniquet proximal to the point at which a large vein is usually present Then a 25 gauge hypodermic needle attached to a small syringe filled with saline is introduced into the small vein and the saline injected against the tourniquet This often dilates the larger vein enough so that it can be entered with a large needle The tourniquet is left in place and the saline injection from the small syringe and needle continued until the larger vein is entered successfully The small needle is then of course removed

INSERTION OF NEEDLE

The technic of repeatedly successful venipuncture is a simple art easily mastered but often neglected by the busy physician A relatively constant and rigid routine produces the best results The tourniquet should be placed 2 5 cm (1 2 in) above the contemplated point of insertion of the needle and secured firmly at a pressure sufficient to occlude the venous return without obstructing the arterial outflow After placement of the tourniquet the available veins are surveyed closely for size distensibility presence or absence of rigidity and especially for direction or course in the subcutaneous tissue When it is deemed advisable to make the injection painless the site of entry of the needle is infiltrated with procaine and the procaine carried deep along the vein wall This helps to prevent vasospasm when the needle approaches or enters the vein

The optimal site of entry of the needle is that point along the course of the vein that permits placement of the needle into the vein under direct observation and its insertion for some distance into the vein and that permits the needle to lie flat on the skin surface for secure anchoring After the site of entry has been determined the syringe with the appro

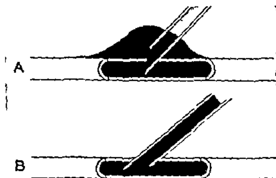


FIG 38 —*A* difficulties encountered with bevel up approach to venipuncture note extravasation and likelihood of puncture of opposite wall of the vein *B* simplicity and safety of introduction of needle with bevel down (From Lundy)

priate needle previously inspected to determine sharpness and absence of burrs is taken in the preferred hand while the other hand grasps the extremity so that the thumb exerts traction on the skin overlying the vein and fixes the vein This is an important element in the technic because fixing of the vein inhibits its mobility and appreciably facilitates its puncture

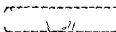
by the needle The needle is then introduced into the skin with the bevel down Figure 38 indicates why the bevel down approach is better On some occasions particularly in large veins the needle may be introduced with the bevel up but incomplete entry into the vein and passage through it are more likely to occur, and once one becomes accustomed to introducing the needle with the bevel down there is little or no need for introducing it with the bevel up

The needle should be introduced into the skin after the point of entry has been moved away from a site directly over the vein (Fig 39) This is done to prevent accidental and traumatic entry into the vein from sudden forward pressure as the superficial layers of the skin are passed through After the needle point has gone through the skin and into the sub cutaneous tissue it is brought back over the vein The syringe is then elevated so that the needle de presses the vein wall and appears to be going directly into it The needle is advanced steadily until blood appears in the syringe (slight negative pressure in the syringe may facilitate this) Immediately on the appearance of blood in the syringe the syringe is depressed so that it lies along the extremity and the needle is advanced in the vein for some distance This is accomplished by first (as pointed out earlier) determining the direction of the vein and following it closely with the needle and by applying a little undulating motion to the needle as it advances With this technic there is little chance for the needle to go through the vein wall

STEP I

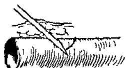


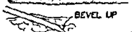
TOP VIEW



STEP II

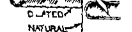
LATERAL VIEW





LARGE VEIN





STEP III



SMALL VEIN



Fig 39.—Venipuncture Method of inserting the needle through the skin at the side of a vein to avoid accidental entry into the vein and possible rupture of the vein wall with extravasation After entering through the superficial layers of skin the needle point is brought back directly over the vein (From Lundy)

It should be remembered that whether the bevel-down or the bevel up technic is used the syringe should be held so that the needle enters the skin and the vein at a position almost parallel with the skin Digging needles into veins with the syringe held at a wide angle with the skin is wont to result in unsatisfactory entry into the vein and passage through it with subsequent hematoma formation

It is essential to proper functioning of an intra venous set up particularly one to be used over a long period to have the needle well into the vein It is poor judgment to stop advancing the needle immediately on its entry into the vein the needle is very likely to be displaced and a good vein is then ruined A needle well advanced into the vein is difficult to displace even with rather active motion by the patient and time is gained by insisting that the needle be advanced well into the vein

It will have been noted that the hand and thumb assigned to grasping the skin of the extremity and fixing the vein were not moved If the course of the vein has been determined during the original inspection and the entry point has been well established according to the routine outlined it is not necessary and it is bad practice to release the vein with the hand and thumb not holding the syringe and attempt to palpate the vein This disturbs the relationships that have been established and interferes with successful and speedy venipuncture

FIXING OF NEEDLE

After the vein has been entered and the needle has been well advanced the portion of the needle remaining outside the skin is secured to the skin with adhesive tape or other suitable fixing material It is not necessary if the foregoing steps have been followed to support the needle with sponges With the bevel down the hub can be placed flat on the skin and with the needle well advanced into the vein it need not be treated with the extreme gentleness and reverence otherwise required

CUT DOWN TECHNIC

On some occasions even with the measures outlined it is difficult or apparently impossible to visualize or palpate suitable veins for venipuncture with a needle It is necessary then to cut down on a vein The vein in the ankle is most often used for this purpose The area is prepared with appropriate antiseptic and a tourniquet placed somewhat higher above the anticipated point of entry than it would be for venipuncture A small incision through the skin is made in a transverse direction down into subcutaneous tissue and a small mosquito forceps is used to separate the tissues in a direction parallel to the course of the vein until the vein can be distinguished The forceps is then passed under the vein so that the point appears on the opposite side and a loop of silk is placed in the tip and brought back under the vein The loop

end is cut leaving two strands of silk After the vein is cleaned of extra tissue, the more distal strand is brought as far back as possible and tied and the ends left long The more proximal strand is gathered in the fingers of one hand and traction upward and cephalward is instituted A small pointed knife blade is then inserted across the top half of the exposed vein to open a slit while leaving the bottom portion of the vein intact The cannula is then introduced into the opening and the upper strand of silk tied around it The wound is then closed with silk and covered with a dressing

Plastic tubing is extremely useful for long standing intravenous administration of drugs and fluids Polyethylene® and Polythene® materials are made into tubing of various sizes which can be introduced into veins in the same manner as cannulas The tubing has the advantage of allowing considerable mobility to the extremity Although there is a tendency to development of fibrous reaction it is perhaps no more than that associated with the use of metal cannulas The tubing can be plugged so that continuous administration of fluids it not necessary to maintain patency

NEEDLES

Lindeman needle —A convenient device for establishing and maintaining an adequate intravenous set-up is the Lindeman needle It consists of a large, relatively blunt cannula through which are inserted two smaller needles

TECHNIC After the vein is located and transfixed in the usual manner a small incision is made over the vein and the device with its component parts is inserted in the vein When the vein is entered



Fig 40—Tocantins needle in place in the body of the sternum

blood runs out in the smallest needle then both small needles are removed and the blunt cannula is inserted well into the vein and secured in place It provides a means for placing a cannula without the necessity of cutting down and isolating the vein surgically

Tocantins needle—Another useful device for the introduction of fluids into the circulation is the Tocantins needle This is a short wide bore needle which has a stilet It is introduced into the marrow cavity of the sternum (Fig 40) the upper end of the

tibia or the lower end of the femur Fluid introduced into these areas enters the circulation directly and in an amount and rate equivalent to those obtained by intravenous administration

TECHNIC The area through which the needle is to be placed is prepared with an antiseptic solution A small nick is made in the skin at the point of entry and the needle with its stilet is advanced steadily and with a boring motion until the point pierces the outer table of bone and springs free into the marrow cavity The stilet is removed and marrow is obtained by aspiration with a syringe to determine the position of the needle point When the position is established the needle is fixed in place by a small clamp on the shaft so that it cannot advance farther into the cavity and if placed in the sternum perhaps penetrate mediastinal structures Note that when placing a Tocantins needle in the sternum one must be careful to avoid inserting it beyond the marrow cavity into the mediastinum

In the sternum the needle is placed usually in the wide flat area just below the junction of the manubrium and body This is the site of entry generally used in adults In children it is sometimes easier to enter the flat surface at the upper end of the tibia or the lower end of the femur

The Tocantins needle is useful when the veins are poor or not accessible When properly placed it is a very stable arrangement permitting protracted intravenous medication without limiting the patient s

movement and with little chance of displacement It is usually necessary when slow drips are being maintained to clean the needle regularly with 5 10 cc of normal saline injected with reasonable force through a syringe connected directly to the needle The mar

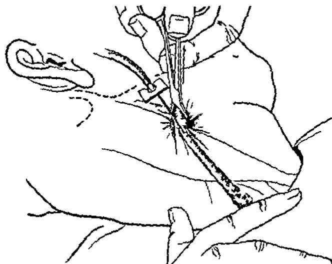


Fig 41—Method of fixing and entering the jugular vein. (From Lundy)

row tends to clog the end of the needle and stop or significantly slow the drip

JUGULAR VEIN TECHNIC

One vein is reasonably accessible but seldom used and sometimes should be considered in emergencies

This is the jugular vein, and it is well to be familiar with a technic for its use The procedure is especially to be considered when intravenous apparatus inserted elsewhere might be in the way of other surgical procedures

PROCEDURE The patient lies on his back with head turned to the side The skin is prepared and an assistant compresses the jugular vein above the clavicle with a finger After appropriate anesthetization with procaine about midway in the exposed neck a towel clip is placed in the skin and under the vein The clip is held in one hand and traction placed on the vein in a cephalad direction With the other hand the needle is inserted in the vein midway between the point of transfixion with the towel clip and the point of compression with the assistant s finger (Fig 41) The needle is then fixed in place in the usual manner

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Drugs and Preparations

NO ACCURATE statement of dosage of drugs can be made that will apply to all conditions The doses listed in the *United States Pharmacopeia National Formulary* and *New and Nonofficial Remedies* are average—that is the average dose found by clinical usage to be safe and effective Such factors as age, size weight individual peculiarities and pathologic state oftentimes require considerable deviation from these average doses The dosages given in this text frequently deviate from the average official doses and are based on the require ments of the situation and the physical state of the patient

The packaging of some of the drugs recommended in the text may not be familiar to the physician Ac cordingly a list is given here with the forms of prep arations Many of the drugs referred to need no special mention Obviously all the sizes of tablets ampules vials etc of the various manufacturers cannot be listed Since various manufacturers may supply the same drug under various trade names the source of supplies is omitted from this edition For a more complete listing

of the packaging we can only refer the reader to *New and Nonofficial Remedies Modern Drug Encyclopedia* and catalog items from the various pharmaceutical manufacturers

Analgesics

Meperidine hydrochloride (Demerol hydrochloride®)
Tablets—50 and 100 mg (3/4 and 1 1/2 gr) each
Ampules—2 cc (50 mg per cc) (3/4 gr)
Vial—30 cc (50 mg per cc)

N allyl nor morphine hydrochloride (Nalline®)
Ampules—1 and 2 cc (5 mg per cc) (1/12 gr)

Levallorphan tartrate (Lorfan tartrate®)
Ampules—1 cc (1 mg per cc)
Vials—10 cc (1 mg per cc)

Anesthetics

Vinyl ether (Vinethene®)
Vials—10 cc
Bottles—25 50 and 75 cc

Tribromoethanol solution (Avertin with amylene hydrate)
Each cc contains 1 Gm (15 gr) of tribromoethanol and 0 5 Gm (7 1/2 gr) of amylene hydrate

Antihistaminics

Diphenhydramine hydrochloride (Benadryl hydrochloride®)
Capsules—25 and 50 mg (3/8 and 3/4 gr)
Elixir—10 mg per 4 cc (1/6 gr) pints and gallons

Tripelennamine hydrochloride (Pyribenzamine hydrochloride®)
Tablets—25 and 50 mg (3/8 and 3/4 gr)
Ampules—1 cc (25 mg per cc) (3/8 gr)
Elixir—30 mg per 4 cc (1/2 gr) pints and gallons

Phenindiamine tartrate USP (Thephorin®)
Tablets—10 and 25 mg (1/6 and 3/8 gr)
Sirup—10 mg per 4 cc (1/6 gr) pints and gallons

Chlorpheniramine maleate USP (Chlor Trimeton®)
- Tablets—4 mg (1/12 gr) each
- Ampules—1 cc (10 mg per cc) (1/6 gr)
- Sirup—2 mg per 4 cc (1/30 gr) pints

Cardiovascular agents

Digitalis preparations A large number of this group of drugs are available and for complete listing see NNR and USP Digitalis leaves and tincture are supplied so that 0 1 Gm or 1 cc shall be equivalent to 1 0 USP digitalis unit

Digitoxin USP
- Tablets—0 05 0 1 0 15 and 0 2 mg
- Ampules—1 cc (0 2 mg) (1/300 gr)
- Vials—10 cc (0 2 mg per cc) (1/300 gr)

Lanatoside C (Cedilanid®)
- Tablets—0.5 mg (1/120 gr)
- Liquid—1 and 2 oz (0 5 mg per cc) (1/120 gr)

Desacetyl lanatoside C (Cedilanid D®)
- Ampules—2 and 4 cc (0 2 mg per cc) (1/30 gr)

Ouabain USP
- Ampules—1 cc (0 1 mg per cc) (1/600 gr)
  1 cc (0 5 mg per cc) (1/120 gr)

Quinidine sulfate USP
- Tablets—100 200 and 300 mg each (1 1/2 3 and 5 gr)
- Capsules—(Same as above)

Quinidine gluconate
- Vials—10 cc (0 08 Gm per cc) (1 1/3 gr)

Erythrityl tetranitrate (Erythrol tetranitrate®)
- Tablets—15 and 30 mg (1/4 and 1/2 gr)

Pentaerythritol tetranitrate (Peritrate®)
- Tablets—10 and 20 mg (1/2 and 1/3 gr)

Papaverine hydrochloride USP
- Tablets—30 60 100 and 200 mg (1/2 1 1 1/2 and 3 gr)

Dextran (Plavolex®)
- Bottles—500 cc

Heparin sodium USP
Vials—10 cc (1 000 units per cc)
10 cc (5 000 units per cc)
5 and 10 cc (10 000 units per cc)
1 and 5 cc (20 000 units per cc)
USP Heparin is standardized so that 100 units is *equivalent to 1 mg*
Depo Heparin® } Ampules—1 cc (200 mg per cc) (3 gr)
Depo repository® }
Hexamethonium (Esomid® Methium chloride® Bistrium chloride® Bistrium bromide®)
Vials—10 cc (25 and 100 mg per cc) (3/8 and 1 1/2 gr)
Procaine amide hydrochloride USP (Pronestyl hydrochloride®)
Capsules—0 25 Gm (4 gr)
Vials—10 cc (10 mg per cc) (1/6 gr)
Protamine sulfate
Ampules—5 cc (10 mg per cc) (1/6 gr)
Vitamin K (Mephyton®)
Tablets—5 mg (1/02 gr)
Ampules—1 cc 50 mg per cc (3/4 gr)
Polyvinyl pyrrolidone PVP (Macrose®)
Bottles—5 000 cc (3 5%)

Central Nervous System Stimulants

Amphetamine sulfate (Benzedrine sulfate® Amphedrine®)
Tablets—5 and 10 mg (1/12 and 1/6 gr)
Ampules—1 cc (20 mg per cc) (1/3 gr)
Elixir (bottles)—5 mg per 5 cc (1/12 gr)
Dextro amphetamine sulfate (Dexedrine®)
Tablets—5 mg (1/12 gr)
d Desoxyephedrine (Amphedroxyn® Desamine® Desoxyn® Drinalfa® Methedrine®)
Tablets—2 5 and 5 mg (1/24 and 1/12 gr)
Elixir (Bottles)—2 5 mg per 4 cc (1/24 gr)

Picrotoxin
Ampules—1 and 2 cc (3 mg per cc) (1/20 gr)

Central Nervous System Depressants (barbiturates)
Barbital
Tablets—0 3 Gm (5 gr)
Phenobarbital (Luminal®)
Tablets—8 15 30 and 100 mg (1/6 1/4 1/2 and 1 1/2 gr)
Elixir (bottles)—15 mg per cc (1/4 gr)
Barbital sodium
Tablets—0 3 Gm (5 gr)
Phenobarbital sodium USP
Tablets—15 30 and 100 mg (1/4 1/2 and 1 1/2 gr)
Amobarbital sodium USP (Amytal sodium®)
Capsules—60 and 180 mg (1 and 3 gr)
Pentobarbital sodium USP (Nembutal sodium®)
Tablets—30 50 and 100 mg (1/2 3/4 and 1 1/2 gr)
Capsules—(Same as above)
Vials—50 cc (50 mg per cc) (3/4 gr)
Secobarbital sodium (Seconal sodium®)
Capsules—30 50 and 100 mg (1/2 3/4 and 1 1/2 gr)
Vials—20 cc (50 mg per cc) (3/4 gr)
Thiopental (Pentothal sodium®)
Ampules—0 5–1 0 Gm (7 1/2–15 Gm)
Make up fresh with distilled water to 2 5 per cent solution for injection

Peripheral Nervous System Drugs
Epinephrine USP (Adrenalin®)
Ampules—1 cc 1 1 000
Vials—30 cc 1 1 000
Adrenalin in oil ampules—1 cc 1 500
Ephedrine USP
Ampules—1 cc (50 mg per cc) (3/4 gr)
l Norepinephrine (Levophed®)
Ampules—4 cc 1 1 000

- Mephentermine sulfate (Wyamine®)
  - Ampules—1 cc (15 mg per cc) (1/4 gr)
  - Vials—10 cc (15 mg per cc) (1/4 gr)
- Methoxyphenamine hydrochloride (Vasoxyl®)
  - Ampules—1 cc (20 mg) (1/3 gr)
  - Vials—20 cc (50 mg per cc) (3/4 gr)
- Phenylephrine hydrochloride (Neosynephrine®)
  - Ampules—2 cc 0 25%
  - Solution—1 and 5 cc 1%
- Paredrine with boric acid
  - Bottles—1%
- Isoproterenol hydrochloride (Isuprel® Norisodrine®)
  - Vials—10 cc 7 100
- Neostigmine methylsulfate USP (Prostigmin methyl sulfate®)
  - Ampules—1 cc 1 2 000
    - 1 cc 1 4 000
  - Vials—10 cc 1 2 000
- Neostigmine bromide USP (Prostigmine bromide®)
  - Tablets—15 mg (1/4 gr)
- Methacholine chloride USP (Mecholyl chloride® Mecholyl bromide®)
  - Tablets—0 2 Gm (3 gr)
  - Ampules—1 cc (0 025 Gm) (4 gr)

Miscellaneous drugs

- Carbon dioxide
  - Available in D cylinders in 100% concentration in mixtures of 5 per cent CO and 95 per cent O and of 10 per cent CO and 90 per cent O
- Corticotropin (ACTH Achar®)
  - Vials—10 25 and 40 units
- Cortisone acetate USP (Cortone® Cortogen acetate®)
  - Tablets—5 and 25 mg (1/12 and 1/38 gr)
  - Vials—20 cc (25 mg per cc)
    - 10 cc (50 mg per cc)
- Botulism antitoxin
  - Vials—10 000 units each

Tetanus antitoxin
  Vials—1 500 3 000 5 000 10 000 20 000 and 40 000 units each

Antivenin (Crotaline species)
  Vials—2 5 cc

Antivenin snake polyvalent (bites by viperine snakes of the United States)
  Package combination—15 cc

Antivenin Lactrodectus mactans (treatment black widow spider)
  Vials—2 5 cc

Bishydroxy coumarin USP (Dicumarol®)
  Tablets—25 50 and 100 mg (3/8 3/4 and 1 1/2 gr)
  Capsules (Same as above)

2 3 Dimercaptopropanol (Dimercaprol® BAL®)
  Ampules—4 5 cc (10% BAL in peanut oil plus 20% benzyl benzoate)

Ethylenediaminetetraacetic calcium salt (Versenate® Disodium calcium)
  Ampules—5 cc containing 1 Gm (15 gr)
  Tablets—500 mg (7 1/2 gr)

Caffeine and sodium benzoate USP
  Ampules—2 cc (0 25 and 0 5 Gm) (4 and 7 1/2 gr)

Diphenylhydantoin sodium USP (Dilantin sodium®)
  Capsules—30 and 100 mg (1/2 and 1 1/2 gr)

Prednisone (Diphentoin® Deltra® Deltasone® Co Deltra®)
  Tablets—1 2 5 and 5 mg (1/60 1/24 and 1/12 gr)

Apomorphine USP
  Tablets—5 mg (1/10 gr)

Chlorpromazine (Thorazine®)
  Tablets—10 25 50 100 and 200 mg (1/6 3/8 3/4 1 1/2 and 3 gr)
  Vials—10 cc (25 mg per cc) (3/8 gr)

Promazine (Sparine®)
  Tablets—10 25 50 100 and 200 mg (1/6 3/8 3/4 1 1/2 and 3 gr)
  Vials—2 and 10 cc (50 mg per cc) (3/4 gr)
Meprobamate (Equanil® Miltown®)
  Tablets—200 and 400 mg (3 and 6 gr)

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